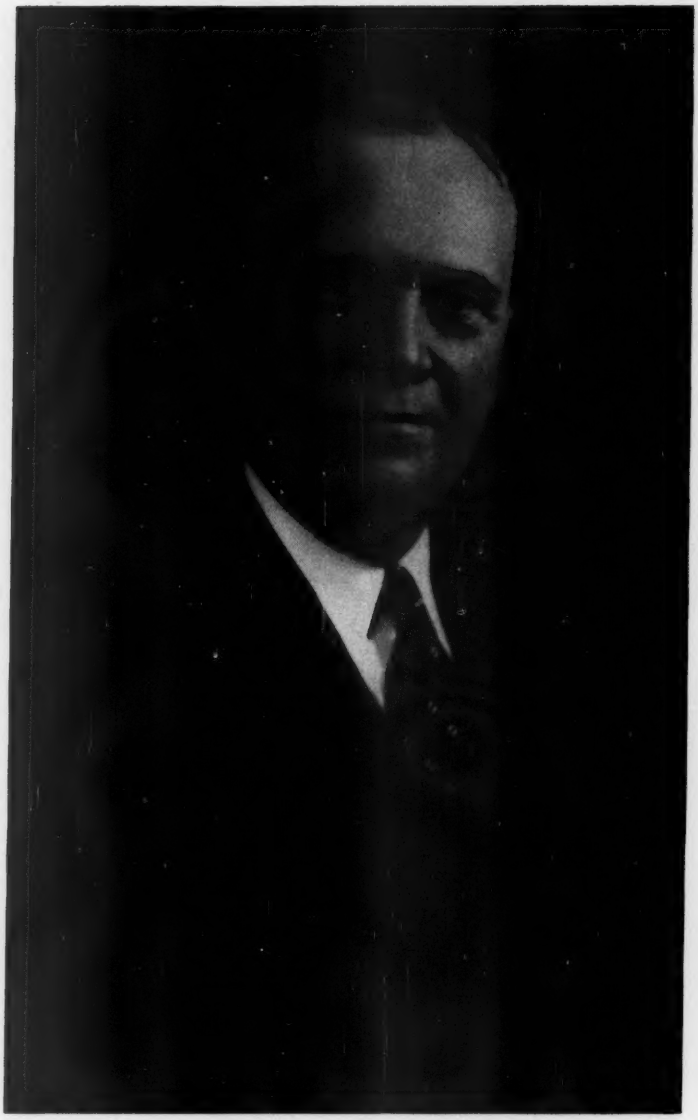

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President of The Southeastern Surgical Congress, 1940.

The Southern Surgeon

Subscription in the United States, $5.00

Vol. IX, No. 3

March, 1940

PREOPERATIVE AND POSTOPERATIVE USE OF DRUGS IN SURGERY OF THE GASTROINTESTINAL TRACT

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FROM a thorough knowledge of the various physiologic functions of the gastrointestinal tract, such as secretion, digestion, absorption, and the maintenance of physical, physiologic, biochemical, and biologic balances, it can be definitely stated that the gastrointestinal tract is the patient's "life line." Without it we cannot survive; with it, but in physiologic imbalance due to a variety of causes, unless the imbalance is corrected we cannot live. It is the latter conditions upon which present-day surgery of the gastrointestinal tract in its broader sense has been founded. Therefore, all the present-day different forms of surgical therapeusis, both mechanical and medicinal, must be based entirely upon physiologic and biochemical principles.

In all surgical interventions of the gastrointestinal tract the first and primary requisite is the preparation of the patient for the operation. Moynihan, many years ago, stated that surgery has been made safe for the patient; now the patient must be made safe for the operation. I would add that both patient and surgeon be made safe for the operation. In order that the patient may be made safe for surgery, it is necessary properly to prepare the patient before operation in order that all functions of the body be brought near the basal physiologic line.

The restoration of the preoperative physiologic balance of the surgical patient and the maintenance of this balance during the op-

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NOTE:—Now that THE SOUTHERN SURGEON is appearing every month, the Editors believe that they are justified in breaking the precedent hitherto obtaining of publishing no portrait of a man during his lifetime. Beginning this year therefore they expect to follow the usual custom of printing the photograph of the President of the parent organization. They regret that they have not been able to publish the picture of each former President of The Southeastern Surgical Congress during his term of office.

eration and throughout the postoperative recovery period may be accomplished by the various means, including drugs, designated in the following outline:

I. PREOPERATIVE

A. Preparation of the patient based on physiologic principles
   1. Water balance
      a. Orally
      b. Subcutaneously
      c. Intravenously
   2. Blood
      a. Corpuscular elements
      b. Electrolytes balance
      c. Blood volume balance
      d. Plasma protein balance
   3. Glycogen balance
   4. Vitamin balance
      a. Vitamins A, B, C, and K
   5. Liver extract
   6. Adrenal cortical hormone
   7. Hydrochloric acid balance
   8. Sulfanilamide
   9. Intestinal evacuants
      a. Purgation
      b. Enemas

B. Anesthetic preparation
   1. Sedation
   2. Basal anesthetics
      a. Subcutaneous
      b. Oral
      c. Intravenous
      d. Rectal
   3. Inhalation anesthetics
      a. Ether
      b. Cyclopropane and oxygen
      c. Ethylene and oxygen
      d. Nitrous oxide and oxygen
   4. Procaine hydrochloride and allied drugs
      a. Spinal
      b. Regional
      c. Local
      d. Splanchnic

II. OPERATIVE

A. Maintenance of physiologic balance during the operation
   1. Water balance
      a. Phleboclysis
   2. Solutions used
      a. Sodium chloride
      b. Dextrose
      c. Ringer's
      d. Hartmann's
   3. Surgical shock prophylaxis
      a. Vasoconstrictors
         1. Epinephrine chloride
         2. Ephedrine chloride
         3. Neosynephrin chloride
         4. Metrazol
         5. Camphor
      b. Blood transfusions
         1. Unmodified blood
         2. Citrated blood

III. POSTOPERATIVE

A. Maintenance of physiologic balance as indicated preoperatively
B. Sedation
   1. Morphine
   2. Dilaudid
   3. Pantopon
   4. Barbiturates
C. Intestinal stimulants
   1. Morphine
   2. Physostigmine
   3. Prostigmine
   4. Hypertonic saline
   5. Procaine hydrochloride
      a. Splanchnic anesthesia
      b. Spinal anesthesia
   6. Choline and derivatives
   7. Pituitary derivatives
   8. Oxygen

Before Operation

Water imbalance is a serious handicap to the patient undergoing a major surgical operation. According to Coller[1], the fluid requirements necessary to maintain normal kidney excretion and prevent water imbalance amounts to 2500 or 3500 c.c. per day. In debilitated and dehydrated patients the requirement may exceed the above amounts. If water imbalance is not corrected preoperatively, the operative and postoperative states increase the fluid requirements to such a degree that the patient becomes dehydrated, blood concentration occurs, anuria develops, and the patient succumbs. The electrolytes of the blood are mainly concerned in the prevention of acidosis and alkalosis. Of these the most important is sodium chloride. All of the electrolytes can be given by using Ringer's or Hartmann's solution, the latter being preferable because of the sodium lactate which is supposed to increase fluid retention. We usually add dextrose in 5 per cent concentration to the above fluids to supply the necessary food. Blood deficiencies usually accompany prolonged and debilitating disease processes occurring in the gastrointestinal tract. The two most important deficiencies are a reduction in the total number of red cells and a decrease in plasma protein. Both of these deficiencies have a definite effect upon the prevention of postoperative complications. In the former, the oxygen carrying capacity of the blood is greatly limited, whereas in the latter, nutritional edema is prone to occur. When the oxygen capacity of the blood is lowered, all tissues suffer different degrees of anoxemia resulting in various imbalances.

In plasma protein depletion, Lapore[2] has shown that in the experimental animal tissue, edema and sodium chloride retention occurs. McCray, Borden, and Ravdin[3] have demonstrated experimentally and clinically that hypoproteinemia produces edema of the gastrointestinal tract as well as a decrease in motor activity with delayed emptying. Thompson, Ravdin, and Frank[4] showed experimentally that hypoproteinemia resulted in disruption of the abdominal wound in 72 per cent of their experimental animals. Therefore, the maintenance of plasma protein balance is absolutely necessary for retention and equal distribution of body fluids as well as supplying the necessary stimulus to sound wound he ling.

The blood corpuscle and hemoglobin deficiencies can be corrected by repeated blood transfusions, and the administration of iron and of liver extract. The correction of plasma protein imbalance can be accomplished by repeated transfusions of whole blood. If hypoproteinemia exists in the presence of normal corpuscular elements, the plasma protein deficiency can be corrected by the intravenous administration of Ravdin's[5] lyophile plasma.

Glycogen deficiency has a very serious effect upon the integrity of the liver parenchyma. According to Ravdin[6], necrosis of the liver occurs in the presence of glycogen depletion. He also stated that in order to protect the liver and prevent necrosis of the parenchyma both the glycogen and plasma proteins must be maintained at normal levels. Dextrose administered either by mouth or by phleboclysis in 5 to 10 per cent solutions restores both liver and muscle glycogen to normal. As stated above, the plasma protein balance is maintained by either blood transfusions or lyophile plasma.

The vitamins play a most important role in surgery of the gastrointestinal tract. They are intimately associated with motor function of the intestine, chronic infections, hemorrhagic diathesis, and wound healing (epithelium and connective tissue). Vitamins A and C according to Anderson[7] have a definite effect on wound healing especially regarding its tensile strength. Wolbach[8] states that in vitamin A deficiencies there occur hyperplasia and keratinization of the epithelial cells. According to this author, vitamin B is concerned more directly with the physiology of the nervous system. It also has an effect on the motor activity of the intestine (typical example is the pellagra patient). In vitamin C deficiencies according to Lanman and Ingallis[9] there is a definite decrease in the tensile strength of surgical wounds. Bleeding tendencies are present in scurvy, a vitamin C deficiency. Marked deficiencies in the production of collagen fibers is noted in vitamin C imbalance.

It has been shown recently that the hemorrhagic tendencies in the presence of jaundice is due to vitamin K deficiency. This tendency to bleed is dependent upon the failure of vitamin K absorption from the gastrointestinal tract. Therefore, almost all of the vitamins play a part in the normal function of the gastrointestinal tract as well as in reparative processes following surgical procedures upon the stomach, small and large bowel. Preoperatively in all debilitated patients with and without associated infections who have surgical diseases of the intestines and who have received insufficient and imbalanced diets, should have large doses of vitamins. All jaundiced patients requiring operations upon the gastrointestinal tract should receive bile salts and vitamin K preoperatively. All of the vitamins can be given by mouth. However, if the oral route is not available they should be given subcutaneously.

Liver extract is indicated in all patients that show varying degrees of blood corpuscle deficiencies. I believe that it is also indicated in cases with liver dysfunction. In both instances the liver extract given subcutaneously acts as a stimulant. Two cubic centimeters of the extract can be given every other day for several doses, obtaining the maximum effect in a few days.

Adrenal cortical hormone is indicated in all debilitated patients as well as those suffering from intestinal obstruction, ileus, and prolonged chronic infections. It is supposed to have a detoxifying effect upon toxins as well as promoting sodium chloride retention. Hydrochloric acid is of value in all cases of achlorhydria and especially in carcinoma of the stomach with pyloric obstruction. In obstruction of the pylorus due either to benign or malignant lesions with diminished or absent hydrochloric acid, the previously sterile stomach becomes a cesspool for bacteria. From both a bacteriologic and surgical viewpoint it is comparable to the sigmoid. Therefore, dilute hydrochloric acid should be given three times a day following lavage of the stomach. The acid will not only inhibit the growth but will destroy a large number of bacteria and make the stomach safe for surgery. Fedoreev[10] recently reported a decrease in mortality following gastric resection for cancer from 40 to 16 per cent. Fedoreev prepared the patient's stomach for surgery by daily lavage with dilute hydrochloric acid. Daily bacterial checks are indicated in this method of preparation of the stomach before surgery is undertaken. In all obstructions, especially those of the small bowel, marked and rapid physiologic imbalances occur. In high intestinal obstruction marked water imbalance, hypochloremia and ileus occur so rapidly that if not relieved by appropriate means death rapidly ensues. The restoration of physiologic balance in acute intestinal obstruction by the administration of fluids containing dextrose and the electrolytes of the blood (Ringer's solution), plasma proteins, and adrenal cortical hormone, the mechanical decompression of the gas-distended and water-logged intestine, and the maintenance of intestinal tone by morphine and prostigmine have materially reduced the mortality. In obstruction of the colon and rectum by new growths besides mechanically cleansing the bowel, Garlock and Seley[11] have advocated the use of sulfanilamide to decrease the bacterial flora. Cultures from the growth and pericolonic tissue revealed the streptococcus hemolyticus and colon bacillus to be the predominating organisms. In twenty-one cases they gave 15 grains every four hours, day and night, for three days before operation. In most of their cases the sulfanilamide was continued for two to three days after operation. In the twenty-one cases so treated the streptococcus was not recovered in any of the cultures made postoperatively. If this preliminary report is corroborated, chemotherapy as a preoperative therapeutic measure is definitely indicated.

In the preoperative use of drugs in surgery of the gastrointestinal tract castor oil, magnesium sulphate, and sodium sulphate as purgatives to cleanse the intestinal tract will only be mentioned in order to condemn them. Alvarez[12], in 1918, condemned both preoperative

and postoperative purgation in the surgical patient. He demonstrated that preoperative purgation was responsible for the development of postoperative distention and gas pains. After twenty years of clinical experience without their use, either preoperatively or postoperatively, I consider them as being entirely unnecessary and definitely harmful.

The use of pre-anesthetic drugs such as morphine and scopolamine, barbiturates and avertin are mainly for the purpose of preventing psychic shock. They are today used as basal anesthetics. I believe that the elimination of psychic shock preoperatively both the night before and prior to the anesthetic by the use of a basal anesthetic has proven definitely valuable in the postoperative recovery of the patient. The various drugs for basal anesthesia can be safely administered orally, subcutaneously, intravenously, and rectally. The anesthetist should be consulted as to his preference of basal anesthetic drugs and the method of administration.

The general anesthetic should be selected by a graduate physician who has specialized in anesthesia. The type of surgical anesthesia must be individualized for each patient, and the one selected should conform to the patient's physical condition and surgical needs.

The anesthetist and surgeon should consult each other regarding the type of anesthesia to be used in each case. They should agree on the surgical anesthesia to be used: general, spinal, regional, or local. I think if we follow this plan consistently, postanesthetic complications would be eliminated to a large extent.

During Operation

The indication for the use of drugs while the operation is in progress is the occurrence of depressed states (shock) either from surgical trauma or loss of blood. The drugs used are those whose action is of a stimulating character. They are used mainly to elevate the blood pressure and to stimulate the respiratory center. The epinephrine group is the one most frequently used—epinephrine, ephedrine, neosynephrin, and metrazol. Epinephrine and ephedrine in peanut oil have a gradual and prolonged effect in contradistinction to their rapid and fleeting effect in other mediums. A recent addition to the blood pressure raising drugs is neosynephrin. Its action is not so rapid and the pressure effect is sustained longer than the above group. Metrazol is used intravenously for its effect upon both the heart and respiratory center. However in large doses they are all dangerous. If the patient is maintained in physiologic balance during the operation by intravenous administration of proper fluids and blood transfusions when indicated, the need for such drugs as the epinephrine group can be eliminated.

After Operation

The postoperative use of drugs is indicated for the relief of pain, cerebral excitement or intestinal atony. If the patient is maintained in physiologic balance by the methods described under preoperative preparation, the necessity for drugs will be limited to the relief of pain. Therefore, if water balance, blood volume, corpuscle elements, and electrolytes, plasma proteins, and vitamins are maintained in their normal relationship the need for drugs will be almost completely obviated. We must remember that in every patient in whom the gastrointestinal tract has been surgically traumatized there exists a physiologic ileus. The degree and duration of this physiologic ileus depends upon the uncorrected physiologic imbalance and the severity of the surgical trauma. If this physiologic ileus is consistently treated in every patient, ileus as a postoperative complication can be eliminated.

The drugs used for the relief of pain are: morphine, dilaudid, pantopon and codeine. If the patient shows sensitivity to morphine one of the other opiates can be substituted.

The drugs used in the prevention and cure of ileus in order of effectiveness are: morphine, prostigmine, oxygen, hypertonic salt solutions, procaine (spinal or splanchnic), choline and its derivatives, pituitary extract.

We[13] have found that morphine administered every four hours is superior to the other intestinal stimulants in maintaining intestinal tone. We demonstrated experimentally that of all the drugs advocated for ileus, morphine and the physostigmine group produced a greater increase in intestinal tone than all the others. This increase in tone was also sustained over longer periods than the other drugs advocated for the treatment of ileus.

Fine[14] has demonstrated that oxygen therapy has a definite effect on maintaining intestinal tone as well as facilitating the absorption of intestinal gases. The oxygen is given either by a special mask or through an intranasal catheter. The amount given should be approximately five or six liters per minute.

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THE SURGICAL MANAGEMENT OF BILIARY TRACT DISEASE

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ADVANCES in the surgical management of gallbladder and biliary tract disease have been so remarkable within the last quarter century that textbooks are obliged to modify treatises on the subject with each edition. The lines of progress have been in many directions: our knowledge of the physiology of the liver, biliary passages and bile have been augmented, the preoperative and postoperative management of patients has been better standardized, technical advances in the operative work itself has enabled recovery in hazardous cases, and morbidity and mortality statistics have been lowered appreciably. It is our intention to recapitulate these advances and to emphasize the role they play in the management of one's patients; in so doing we are presenting a series of personal cases to exemplify their clinical application.

PHYSIOLOGIC CONSIDERATIONS

The foundation of good results in the management of biliary tract disease is a modern knowledge of the physiology of the liver. One of the most important functions of the liver is concerned with the storage of dextrose as glycogen and releasing it again into the blood stream when demanded. Hepatectomized animals die after a few hours with a rapid fall in blood sugar. If glucose is given intravenously, death can be delayed as much as 24 hours. On the other hand, if as much as seventy per cent of the liver is removed, the remaining portion will not only carry on the demands of the animal but will have regenerated the excised portion within a few weeks.

Another of the functions of the organ is concerned with protein metabolism. Amino acids find their way to the liver by way of the portal vein. Some of them are converted to glucose and urea through intermediate products of lactic acid and ammonia respectively. Others are utilized as such and some investigators maintain that the protein molecule is resynthesized within the liver. After hepatectomy the blood urea level falls progressively provided renal function is not impaired. With bilateral nephrectomy, the blood urea level remains unchanged.

The liver is concerned with other important metabolic processes to which we shall refer from time to time: namely, mineral metabo-

lism, bile pigment metabolism, fat metabolism, the formation of fibrinogen and other essential clotting elements, detoxification of certain poisons and drugs, and the regulation of the plasma-cell ratio in the blood. With regard to the latter, febrile states reduce plasma concentration in the circulation, but this does not occur after denervation of the liver. The detoxification of poisons and drugs is a chapter in itself and we shall refer the reader to the experimental studies concerning phosphorus, barbiturates, salicylates, uric acid, benzoic acid, allantoin, galactose, lactic acid, strychnine, nicotine, polypeptides, quinidine, cinchophen, and many others.

The reticulo-endothelial system has within its ramifications in the liver a highly specialized cell in the so-called star cells of Kupffer. It is here that bilirubin is formed from the breakdown of hemoglobin. This is not the only source of bilirubin for it is also elaborated by the reticulo-endothelial cells of the bone marrow and spleen. The bilirubin is excreted in the bile together with cholesterin, bile acids and bile salts. Animals who are deprived of their bile through biliary fistulas die within a few months with abnormalities of the bones associated with a loss of inorganic constituents. Their stools become fatty and clay-colored and an excessive putrefaction develops with the ensuing diarrhea. If the spleen is removed in such animals, death comes much sooner, apparently from a reduction in hemoglobin. Bile as it is formed in the hepatic lobule differs from that found in the gallbladder where it is stored; the former is thinner and light yellow in color while the latter has a heavier specific gravity and a dark green color. Moreover gallbladder bile contains more base, more bile salts, more calcium, and is definitely acid in reaction, while liver bile has more carbonate, more chloride, and is alkaline in reaction. The flow of bile acids may be augmented mainly by the administration of bile salts and to a less extent by secretin, a hormone (Ivy) elaborated in the duodenal mucosa. Bile not only lowers the acidity of the gastric juices but it is necessary in emulsification of fats in digestion and in activating steapsin, the lipolytic enzyme of the pancreas. Jaundice occurs when the balance between bilirubin formation and bilirubin elimination is disturbed. This comes about in two ways: in the retention type, bilirubin is bound up with the plasma proteins producing an indirect Van den Bergh reaction as we see in congestive heart failure, lobar pneumonia, and hemolytic jaundice. Such bilirubin is not excreted by the liver, and there is a concomitant cloudy swelling of the kidneys preventing its renal excretion. In the obstructive form, the bilirubin is excreted by the bile canaliculi, but due to the back pressure exerted by obstructions of the duct in lithiasis, inflammation and neoplasia, it cannot reach the intestine. The bilirubin thus formed remains uncombined and produces a direct Van den Bergh reaction which

can be eliminated from the kidneys as urobilinogen or unchanged bilirubin.

The release of bile into the duodenum is controlled by the sphincter of Oddi which surround the ampulla of the common duct at its terminal portion. The sphincter will withstand a pressure of 300 mm. of bile without relaxing when there is no digestion taking place. However, when the pressure reaches 50 to 70 mm. the valves of Heister in the cystic duct relax and allow the bile to enter the gallbladder. The gallbladder will in turn develop a pressure as high as 300 mm. but only 100 mm. is necessary to cause the sphincter of Oddi to relax. This would not indicate that there is a reciprocal innervation which relaxes the sphincter when the gallbladder contracts.

The functions of the gallbladder are those of absorption, contraction, and secretion. Bile is formed normally in amounts from 500 to 1000 c.c. daily. After storage in the gallbladder it is concentrated from 500 to 900 per cent by removing water and inorganic salts. Any inflammatory process of the mucous membrane may inhibit its powers of absorption and concentration. Papillomas of the gallbladder may increase this power by increasing the mucosal surface. Riegel and his associates have found that the gallbladder will absorb water much more quickly than inorganic or bile salts, and Ravdin found that sodium chloride and potassium iodide were absorbed promptly. The most potent stimulant to gallbladder emptying is egg yolk. Fatty substances also exert this influence to a less extent and the natural means for its contraction is provided in a hormone of the duodenal mucosa. Extirpation of the gallbladder results in the adoption of its functions by the bile duct system. Hepatic bile is concentrated within the ducts and released as needed for digestion.

Pathologic Considerations

Cholecystitis and hepatitis are diseases peculiar to man. Either can be produced experimentally in animals by the intraperitoneal introduction of Dakin's solution. Judd pointed out that cholecystitis is a local organ manifestation of a disease which affects the whole biliary tree. Any consideration of a patient with impairment of the function of the gallbladder must entail an investigation of the biliary ducts, common duct and pancreatic ducts. Infection of the gallbladder reaches the liver and pancreas through the lymphatics or directly through the biliary tree. Infection of the biliary tract produces stasis and obstruction, and with a deranged cholesterin metabolism, there results the nucleus for gallstone formation. Frequently one sees fine deposits of cholesterin in the submucosa of the gallbladder which are particularly frequent during pregnancy when the blood

cholesterol is high. Gallstones may be present without infection of the gallbladder. Cholelithiasis is noted in 50 per cent of the cases of chronic or hemorrhagic pancreatitis, cyst of the pancreas, and in chronic hemolytic jaundice. Moreover, cholecystitis may be produced by injection of pancreatic extract into the gallbladder. When one cultures gallstones, organisms are recovered in about 30 per cent of cases. Cultures of bile are positive in 15 per cent of the cases, cultures of the gallbladder wall in 48 per cent, and cultures of the adjacent lymph nodes in 80 per cent. Thus, many avenues are open for elaboration of theories of the origin of cholecystitis.

Diagnosis

In arriving at a diagnosis of cholecystitis with or without associated biliary tract disorders, we rely on four important criteria. Fair, fat and forty is no longer the axiomatic triad; we see more patients in the second and third decade and again in the sixth and seventh decades than in the former age group. We are cognizant of the many adjuncts of the laboratory together with the vast symptomatology which may be attributed to biliary dyskinesia. We do not feel that any of them is diagnostic without one of the four manifestations.

1. *Pain.* Results of surgery in biliary disorders is apt to be discouraging when pain is absent. These patients complain of pain following the ingestion of a meal rich in fried or fatty foods. The pain is usually in the right upper quadrant and frequently radiates to the shoulder and back. The history of pain frequently patterns that of peptic ulcer with its periodicity, seasonal variation, even to the point of relief with the taking of food or soda. Needless to say, diagnosis rests on the failure of the roentgenologist to demonstrate an ulcer niche, together with other informative diagnostic aids. If calculi lodge in the ampulla, actual ulceration of the duodenum may take place with both syndromes of ulcer pain and biliary tract pain present. The pain is usually due to the spasm of the sphincter of Oddi which has been initiated by the irritation of a calculus, inflammation or neoplasm. Our attention has been called to the type of colic which simulates angina pectoris where removal of the gallbladder brings relief of pain. We are ever conscious of the reciprocal observation when coronary disease produces epigastric pain, and not a few cardiac patients have parted with their gallbladders to no avail. McGowan, Butsch, and Walters have found nitroglycerin as an aid in differentiating biliary tract pain from that arising from other sources. Morphia increases the spasm in the sphincter, raising the intraductal pressure and thereby augmenting the pain; nitroglycerin affords prompt relief as a rule. Sometimes a pericholecyst-

itis will have produced adhesions to the colon or small bowel with symptoms of obstruction. The degree of pain may be unreasonable in cholecystitis. It is interesting to note the number of patients in whom little is found at the operating table. Again, there are those who experience little or no pain and in whom the pathologic process is advanced. In this regard we call attention to the so-called "silent stones."

2. *Tenderness under the right costal margin.* Tenderness is significant only when pronounced. It may be accompanied by jaundice but the presence of the latter is not diagnostic; it merely places one on his guard and forces him to rule out the biliary tree in the differential diagnosis. Deep pressure in eliciting tenderness may be misleading due to the proximity of other important viscera in the region, namely the hepatic flexure of the colon, the duodenum, and the right kidney.

3. *Cholecystograms.* When roentgenograms are positive, they are diagnostic alone in 95 per cent of the cases. Along with positive roentgenographic evidence, we see another 18.5 per cent in whom definite disease is found at operation, but who have been reported with normally functioning gallbladders. When the history of pain is lacking, we have come to put a lot of faith in a negative cholecystogram. What we want to emphasize particularly is that the roentgenogram does not always localize the part of the biliary tree affected just bcause the dye was improperly concentrated. It does not often portray those cases in which malignant change has taken place, and the surgeon should be ever mindful of this in undertaking exploration.

4. *Laboratory adjuncts.* We refer particularly to the Van den Bergh reaction which may be significant of surgical disease especially when blood bilirubin values are high. There are many other laboratory aids namely, galactose tolerance test, bromsulphthalein liver function test, the Quick hippuric acid test, quantitative fibrinogen studies, quantitative blood iodine, a study of clotting properties, estimations of the bile in the urine, urobilinogen, analysis of the stool, icteric index, blood amylase, blood smears, and many others. There are some who rely considerably on the Lyon-Meltzer test where bile is recovered from the duodenal tube and a diagnosis made from a study of its chemical properties. Although this test is helpful to some degree, we do not rely on these findings alone. A study of the properties of the bile is quite essential, however, in the postoperative management of some cases.

Selection of Patients for Operation

In advising operation for biliary tract disease, we take the stand that all stone cases are surgical if the condition of the patient is

satisfactory. Even if there are no symptoms they frequently give trouble later in life when the condition of the patient may be worse. We also advise operation in cholecystitis where there is some doubt concerning the diagnosis. Moynihan once said that he always advised surgery because he did not believe any man was infallible in his diagnosis. In recent years a great deal of discussion has arisen concerning operation in the acute phases of cholecystitis. Totten reported a mortality of zero when he operated within the first three days, whereas his total mortality was 13 per cent. We advocate early operation, and believe that the risk is less if done before forty-eight hours. In delayed acute cases, we rely upon clinical improvement of the patient together with an approach to normal in the leukocytic and sedimentation responses before advising exploration.

In order to determine hepatic function and to have an index of safety of operability, we employ the Quick liver function test. Like Boyce, we believe it forewarns us of those patients who are subject to fatal complications allied with "liver death." This entity was first brought to our attention by Heyd in 1924. Occasionally, after a seemingly simple operation on what was considered a good risk patient, death resulted without apparent warning. The exitus came about in one of three ways. The first group died after a few hours with high fever, lowered blood pressure, and a hepatic insufficiency in a somewhat related anaphylactic death. The second type died after a period of days with anuria due to renal failure, where only cloudy swelling could be demonstrated in the kidneys and liver at necropsy. The third group was an intermediate type of the other two. These patients all had a low hepatic reserve and a direct relationship has been experimentally shown between the liver and kidneys when death comes about through other apparently related causes, namely, thyroid crisis, intestinal obstruction, burns, and adrenal insufficiency. The Quick test has been shown to produce no burden on a damaged liver and give us an index as to the reliability of the liver's detoxifying powers.

Elderly patients stand major procedures well, provided the cardiovascular-renal reserve is adequate. Hypertension per se does not add to the gravity of the prognosis. McQuiston showed conclusively that hypertensives did as well as hypotensives or those with normal blood pressure, provided, of course, there were no vascular changes. Brooks reported a series of these patients in whom the operative mortality was 10 per cent.

Preoperative Preparation

1. *Glucose.* Since the liver is the chief source of available glucose in the form of readily assimilable glycogen, and inasmuch as the

ordinary laparotomy reduces liver function by as much as 25 per cent when no glucose is given preoperatively, we are particularly anxious to supply an ample quantity to gallbladder patients. Althausen showed how the liver could be literally forced to take on a store of glucose, and Coller and Jackson recommended the dextrose tolerance test as a guide in determining the liver's carbohydrate metabolic powers.

2. *Fluids.* A great many of these patients are in negative water balance on admission due to fever, vomitus and diminished intake. Like Coller and Maddock, we advocate a daily intake of 3500 to 4500 c.c. to carry on normal excretion. One should be governed by the urinary output in administering fluids. Ordinarily, we try to enforce a renal excretion of 1000 to 1500 c.c. daily.

3. *Transfusion.* Many of these patients are anemic. Not only does fresh blood meet the demands of their inadequacy but may furnish other mineral and nutritional properties which may be lacking.

4. *Bile and vitamin K.* The bleeding in jaundiced patients has been laid to a prothrombin deficiency. The administration of bile and vitamin K obviates this prothrombin reduction. When the clot of these patients is examined, it appears large and boggy and does not retract properly. This is due to the retention of portion of the serum, and such a clot is inefficient in stopping hemorrhage since it allows the blood to seep through slowly.

5. *Oxygen.* Judd emphasized the value of administering oxygen to patients with jaundice. It not only increased the oxygen saturation curve of the blood with increased oxygenation to the liver cell, but frequently prevents pulmonary complications. Schweigk has shown that decholin (dehydrocholic acid) will also increase the blood flow through the hepatic artery and hence increase the oxygen saturation curve. Winfield obtained similar results with dessicol. This results in a choleresis and diuresis which may or may not be of value in inflammatory states. We do not administer decholin when there is an obstructive lesion to the duct system.

Operative Technic

When the surgeon takes these patients to the operating room, he must be prepared to examine not only the gallbladder, but the liver, ducts, pancreas, spleen and appendix as well. We have used the Kocher-Mayo incision routinely as it gives adequate exposure to all structures pertaining to the biliary system. An advanced degree of hepatitis or cirrhosis may change the plan of operation entirely. In hepatitis, the liver appears swollen, smooth and green-

ish-yellow with a rounded presenting edge. Biliary cirrhosis, on the other hand, presents an atrophic, scarred liver with a granular surface. Varying amounts of ascites may be present. Patients who exhibit considerable liver damage are apt to have a stormy convalescence and the surgeon must undertake to do the surgical procedure which will afford the least trauma and the most adequate method of drainage. Gallbladder surgery has metamorphosed from an era of simple lithotomy by Robbs in 1867, through the two-stage drainage operation of Kocher and the one-stage cholecystostomy of Sims in 1878, to the first cholecystectomy by Langenbuch in 1882. Even so, cholecystostomy still held full reign until about 1915 when it was noted that many of these patients returned for secondary operations. We now prefer cholecystectomy whenever possible. Regarding the technic of removal of the gallbladder, we believe that the retrograde method has been fairly well standardized. In this way the cystic duct with its possible congenital abnormalities may be well isolated and the danger of hemorrhage from neglect to the cystic artery is obviated.

There are but few indications where simple drainage is employed. When the inflammatory process is so acute that the gallbladder cannot be safely shelled from its fossa, or when the nature of the obstruction is such that the gallbladder may be needed for short-circuiting procedure to the stomach or duodenum, cholecystostomy is used. Otherwise its resultant morbidity is 155 per cent higher than the expected rate according to Dublin. We advise gradual decompression in cholecystostomy as suggested by Ravdin to prevent sudden hyperemia of the liver with possible failure.

When the operator finds a dilated common duct, exploration of the duct is mandatory. Oftentimes one will find the same inflammatory process in its mucosa as found in the gallbladder. The stasis and infection in the biliary tree accounts for the pain that frequently persists in noncalculous cases when drainage of the duct is not instituted. A scoop passed upward into the hepatic ducts and downward into the duodenum will rule out duct stones or strictures. In choledochostomy, a gradual decompression is the rule again. One should always palpate the pancreas in these cases. If pancreatitis is present, it will have a firm consistency but not the irregular induration that goes with malignancy. At times a definite diagnosis cannot be made. Drainage of the common duct is the most effective treatment known for inflammatory lesions of the pancreas and severe grades of hepatitis, a good proportion of which are secondary to inflammation or obstruction of the duct. Walters has employed a radio-opaque substance at the operating table to visualize the biliary tree and reveal any evidence of doubt concerning stones. We advise opening the common duct for ade-

quate exploration when indicated, rather than to do an exploration through the cystic duct which may be inadequate. Drainage of the peritoneal cavity is almost always done. Only in a single case have we closed the abdomen without drainage.

Postoperative Measures

Postoperatively these patients receive a continuation of their preoperative measures. Particular attention must be given to adequate amounts of glucose and fluids. We employ transfusion and oxygen without delay if there is the slightest indication. We pay particular attention to the lungs and utilize measures to prevent atelectasis and pneumonia. The Trendelenburg position, carbon dioxide inhalations, intratracheal suction and even bronchoscopy have been used.

Complications

Judd reported the complications of biliary surgery. Death was due to pulmonary causes in 38 per cent, peritonitis in 13 per cent, liver or kidney failure in 13 per cent, hemorrhage in 11 per cent, liver abscess in 8 per cent, pancreatic abscess in 3 per cent, and other causes including shock in the remaining 7 per cent. Our present day preoperative preparation has reduced the dangers of hemorrhage and liver failure to a large degree, and certain postoperative measures have lowered the mortality from pulmonary complications. When drainage through a T-tube has been employed, and when other lesions have been attacked beside the gallbladder, the mortality has been higher. Complications may arise from the too early removal of the T-tube. Payne has shown that the chemistry and physical properties of bile is a good criterion for removal of the tube. Other investigators have made use of a study of intraductal pressures in determining when to remove the tube. We have found that injection of the T-tube with radio-opaque oil is of value in determining if there is any obstruction in the duct.

Results

In our personal series of sixty-seven cases, cholecystectomy was performed in sixty-five, cholecystostomy in two. The common duct was opened and drained in ten cases. Intestinal anastomosis of one form or another was done concomitantly with the gallbladder operation in four patients for conditions related to ulcer, duodenal dilatation or intestinal obstruction. Two patients who were cholecystectomized had previous cholecystostomies without relief, and one patient had a previous cholecystectomy with evidence of a common duct stone remaining.

In the two cases in which drainage of the gallbladder was employed, the indications were those of severe acute cholecystitis and hepatitis in a poor risk patient and liver abscess in the other. Drainage of the common duct was carried out where it was dilated, where there was evidence of pancreatitis, or where there was a severe grade of hepatitis. Of the ten patients so treated, eight had severe hepatitis, three had common duct stones, seven had cholangitis, five had pancreatitis, and three had cirrhosis.

In seven of the cases there was acute cholecystitis; in three of these the gallbladder was gangrenous and perforated; in the other three acute empyema of the organ was present. Other pathologic processes found were of interest; there was 1 case of hydrops, 2 of papilloma, 1 of adenoma of the fundus, 1 of gallbladder, 1 chronic empyema, 1 cholecystduodenal fistula (spontaneous), 10 of cholesterosis, 5 with pericholecystitis, 21 of stones in the gallbladder, and an additional 33 with minor grades of hepatitis.

Of the uncomplicated cholecystectomies the mortality was 3.9 per cent. The gross mortality for the group with and without complications was 7.5 per cent. In the seven acute cases subjected to removal of the gallbladder, there was one death. The causes of death in the series were as follows: hepatorenal failure, 2; pulmonary embolism, 2; sepsis, 1. Of the patients surviving, 32 considered themselves cured; 18 were greatly improved with no residual pain; 5 were improved but had occasions of distress unrelated to the biliary tract, and 3 were untraced. Of the 5 whose results were mediocre only one was found to have stones.

Conclusions

1. Surgery should be employed in patients exhibiting biliary tract disturbances who received little benefit from medical regime.

2. The presence of stones or the suspicion of malignancy demands surgery.

3. One should keep in mind the physiology of the biliary system in preparing his patients for operation; we refer specifically to certain liver function tests which will evaluate surgical risk in many cases.

4. Cholecystectomy is the operation of choice. Drainage of the common duct is imperative where there is cholangitis, duct stone, pancreatitis, or severe grades of hepatitis.

5. In cases of acute cholecystitis seen within the first 48 hours, the results of cholecystectomy are excellent.

6. The results of biliary tract surgery are exemplified in a series

of sixty-seven personal cases wherein the gross mortality was 7.5 per cent.

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MARCH FOOT

A Personal Experience

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MEDICAL papers are commonly based on a physician's experience garnered from his patients, from the literature, and occasionally from both. The experience in the following dissertation, however, was painfully and personally gained by the author himself. The historical data and numerous theories of etiology have been selected from the literature, but when dealing with the clinical entity itself, I know what I am talking about!

March foot is a peculiar, painful condition of a metatarsal, usually leading to spontaneous fracture. The first reference to this condition in the literature is credited to Breithaupt, a Prussian military surgeon who described this painful lesion in 1855 and termed it "Fussgeschwulst." Weissbach again described it in 1877. Neither of these men, however, was certain of the pathology. During the nineties and around the turn of the century, numerous articles on the subject appeared in the German and French literature.

It is surprising that with all of the European reports not a single paper on march foot appeared in English until Jansen made his report before the British Orthopedic Association in 1926. Surprising also is the fact that only eleven years ago was the first case reported in the American literature by Goldman of Montreal. It was not until seven years ago that the first case of march foot occurring in the United States made its appearance in the literature[20].

Etiology

Pauzat in 1887, and others even later, felt that the condition was a periostitis. It was not until 1897 that the possibility of a fracture was considered by Brusquet. The next year Stechow actually demonstrated a fracture of the metatarsal by the miraculously new phenomenon of the x-ray.

In 1899 Kirschner reported 82 cases of march foot. He was the first to describe the condition in a civilian. It is interesting to note that previous observers found the condition only in soldiers, usually after long marches with heavy packs, and most often in new recruits. It is because of this association that the disease has become known as "march foot" or "march fracture." Overstrain in a weak foot has remained the popular concept as to the manner of production. However, Rider said that overfatigue could hardly be the main cause for no case has been reported in a marathon dancer.

Meiser stated that an elongated metatarsal causes a weak foot. The longer the metatarsal the weaker the foot, and the more likely the occurrence of march foot in the elongated bone. Dodd argued that the shorter the first metatarsal the longer the other metatarsals by comparison and hence the weaker the foot. Among the more bizarre etiologic factors may be listed: starvation[9], trophoneurotic elements[22], high heels[2], overweight[6], secondary circulatory changes in the bone due to spasm of the interossei[10], or due to flat feet[17].

Incidence

That march foot has been rarely recognized in this country is shown by the scarcity of papers on the subject. Some idea of its incidence here can be gained by Meyerding's report last year of ten cases seen at the Mayo Clinic. In Europe, however, Bahr was able to collect over 2146 cases as early as 1913.

Men are predominantly affected, but the condition is by no means confined to the male sex.

March foot is typically an affliction of adult life. I have not seen a report of its occurrence in childhood.

The order of frequency of the bones involved are shown by Tobald's report of 228 fractures. Of these the second metatarsal was involved in 112, the third in 98, the fourth in 17, the first in one. March foot affecting the fifth metatarsal has been reported, but in this site it is even rarer than in the first.

Double fractures in the same metatarsal are occasionally reported. Fractures of more than one metatarsal have been seen by Speed and Blake, and Maseritz. Recurring fracture at the same site is rare.

Signs and Symptoms

With no history of trauma and seldom one of overstrain, march foot commences rather insignificantly with a little pain or soreness in the anterior part of the foot. As this progresses inspection shows a puffy swelling entirely limited to the dorsum of the foot near the base of the toes. The swelling pits slightly upon pressure and there is deep tenderness. The skin is shiny, tense and reddened. Symptoms vary in intensity from those so slight as hardly to be noticed, to those severe enough to incapacitate the individual. The condition improves with rest, but resumption of use of the foot aggravates the symptoms. A definite limp is usually present. After fracture has occurred the patient cannot stand with his weight on the ball of the affected foot. The swelling gradually becomes more localized and after several weeks a firm tumefaction of the bone can be made out.

Early x-rays show nothing abnormal. After one to three weeks a hazy periosteal reaction begins to make itself evident around the shaft of the metatarsal. Close inspection may show a fine fracture line at this time. Later, the periosteal shadow becomes more dense, and the fracture more distinct. There is seldom, if ever, appreciable displacement of the fragments. Often the fracture line never becomes definite. One of the most outstanding characteristics of march foot is the overabundance of callus for the small size of the bone and the minuteness of the fracture. The denser the callus becomes the more elongated and spindle-shaped it grows. The fracture gradually unites and after several months the callus becomes still more solid and even tubular. Within six to nine months there is no remaining evidence of disease save a slight thickening of the cortex at the site of fracture.

Diagnosis

There are three conditions that have been confused with march foot: sarcoma, syphilis, and tenosynovitis. Meyerding and others have had to resort to biopsy to differentiate the condition from sarcoma. Straus' case had a previous diagnosis of sarcoma elsewhere and amputation of the leg had been advised. Straus could obtain no history of trauma, and no fracture line was discernible. His patient gave no history of lues and her Wassermann was negative, so he did what he considered safest; removal of the entire second metatarsal. The suspicion of sarcoma of the bone was so positive in one of Dodd's cases that the foot was amputated. Serial x-rays and the history of previously overtaxing the feet, though it be weeks before, should eliminate this horrible error.

Tenosynovitis is more apt to give crepitation upon palpitation and no history of trauma of any kind. The x-rays in tenosynovitis remain negative.

Obviously syphilitic periostitis can be ruled out by history and serologic tests.

March foot will be readily diagnosed if it is only kept in mind.

Treatment

Where possible, march foot can best be treated by rest and elevation. Bed rest is most efficient, crutches next. If neither of these is feasible a plaster cast with walking caliper affords a large amount of rest to the foot. If this is not practicable, weight can be kept off the ball of the foot by having the patient walk on the heel. When walking in this manner and reducing it to a minimum, satisfactory symptomatic relief and healing can be obtained. A new shoe with stiff sole will afford a good deal of immobilization.

Prognosis

The outcome of march foot is uniformly good.

Report of Case

One who is descended from rural Louisiana stock necessarily inherits some instinct for hunting, no matter how meager it may be, and he is apt to try to crowd a six weeks hunting season into the opening day.

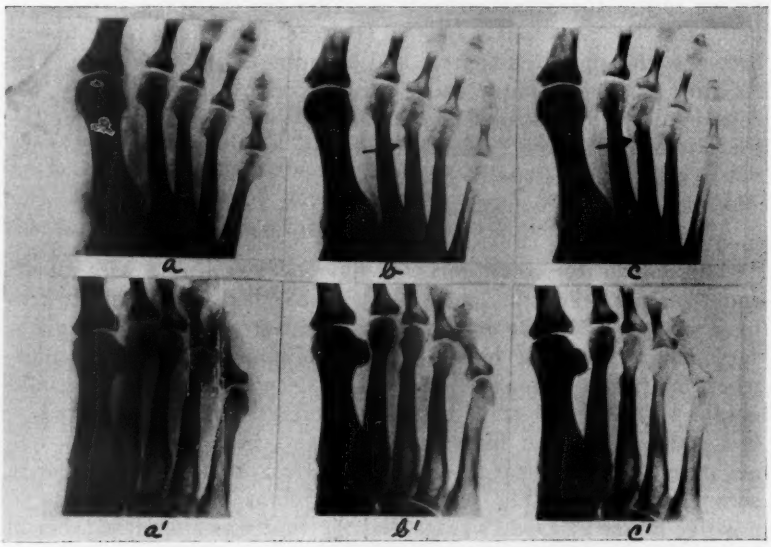

Figure 1. a, Negative roentgenogram 8 days after onset of symptoms, anteroposterior view. a', oblique view. b, Roentgenogram 21 days after onset, note slight periosteal fuzziness, anteroposterior view. b', oblique view. c, Roentgenogram 27 days after onset; anteroposterior view; note increased amount and density of callus. c', oblique view.

My first quail hunt of last season was held in Alabama on Sand Mountain. The date was Nov. 28, 1938. My host was a lean, lanky mountaineer well over six feet tall. Before the day was over I felt his height was mainly composed of legs. Sand Mountain is aptly named, and it is all loose sand. After walking from 5 o'clock in the morning until dusk, it became vividly apparent that locomotion through sand was somewhat different from walking over the floor of a hospital. A dozen quail, however, was ample reward for my extreme fatigue.

On December 20, the opportunity for a repetition presented itself and advantage of it was promptly arranged. The hunting that day was not as hard or as long. We stopped after nine hours, but my feet burned and ached more than previously.

Eight days later, I noticed a little pain in the middle of the ball of my right foot, especially upon walking. It was so slight that I did not inspect the foot. This pain gradually increased in intensity and four days later I examined the foot for the first time. There was a little swelling on the dorsum just

proximal to the base of the third toe. The swelling pitted slightly on pressure and there was tenderness deep in. The skin was glazed and slightly reddened. X-ray that day was negative (fig. 1, a and a′).

For two more weeks the symptoms slowly progressed and I developed a slight limp. X-ray at that time showed a periosteal fuzziness around the distal end of the shaft of the right third metatarsal (fig. 1, b and b′).

When one more week had elapsed, and the x-ray showed marked growth of the shadow, but no fracture line (fig. 1, c and c′), I began to envisage a

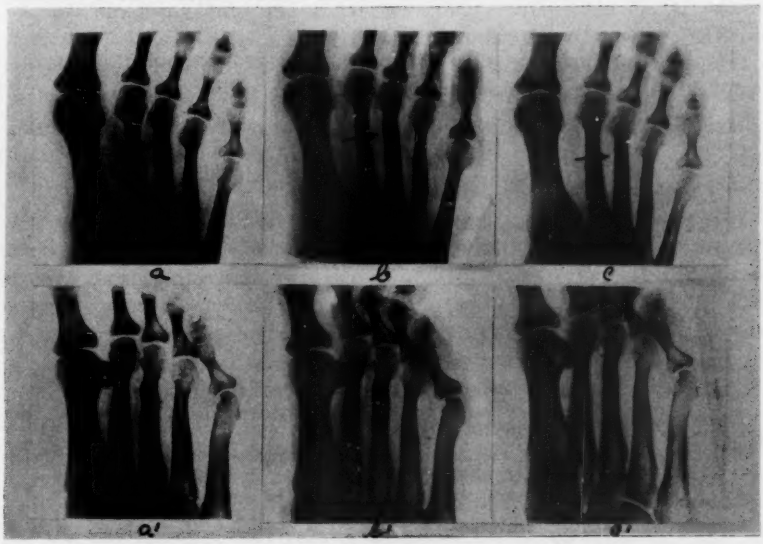

Figure 2. a, Roentgenogram 43 days after onset of symptoms; anteroposterior view; note dense spindle-shaped callus. a′, oblique view. b, Roentgenogram three months later; anteroposterior view; note hard callus almost tubular in shape. b′, oblique view. c, Roentgenogram eleven months after onset; anteroposterior view; note only a little thickening of the cortex at site of fracture. c′, oblique view.

cork leg. I forthwith wrote my former teacher, Dr. Henry W. Meyerding, "I believe I have march foot but I would feel much more comforted if I could see the fracture line." And I did feel relieved when he wrote that he could see the fracture line.

As the pain and swelling increased, so did my limp. In walking, intentional weight-bearing on the heel of the affected foot soon became automatic. I found that a new pair of shoes with extra thick soles gave my foot much comfort by increased support and immobilization.

The callus was much denser and spindle-shaped when x-rayed two weeks later (fig. 2, a and a′), and symptoms were gradually subsiding. After three more months the foot was free of pain and the callus was very hard and almost tubular (fig. 2 b and b′). Eleven months after onset, x-ray revealed nothing but a residual thickening of the cortical bone at the site of fracture (fig. 2 c and c′).

Last month I demonstrated that healing was complete by two hard days of hunting in Minnesota. My only casualties were blisters on the heels from new shoes, but the ring neck pheasants in the bag more than compensated for these.

SUMMARY

1. Spontaneous fracture of a metatarsal can occur after long, unaccustomed walking.

2. March fracture probably occurs much more often than is recognized.

3. The condition is easily differentiated from tenosynovitis and syphilis, less easily from sarcoma.

4. Conservative treatment renders uniformly excellent results.

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LEFT PARADUODENAL HERNIA

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PARADUODENAL hernias are located about the duodenojejunal flexure. They are relatively uncommon and may be found at any age. Characteristically, they are composed of small intestine protruding through a congenital or anomalous opening which is entirely within the abdomen. Paraduodenal hernias are classified as right or left. Fifty-one right and more than one hundred left have so far been recorded in the literature.

In 1776, Neubauer[1] described the first case on record, but it was not until 1857 that Treitz[2] enunciated the first clear conception of the pathogenesis of these hernias. Treitz described the various fossae located about the duodenojejunal junction. He felt that these hernias were congenital and that they formed because of failure of complete fusion of the peritoneal leaves during the process of rotation. He further believed that these were widened and deepened by the pressure and peristaltic movements of the intestines thus producing a true hernia. Waldeyer[3] thought that the elevation of the peritoneum by blood vessels, namely the inferior mesenteric vein and the left colic artery, was the important factor in their formation. These potential hernial sacs were considered by Treves the remains of the early mesoduodenum. Moynihan[5] listed nine fossae situated about the duodenojejunal junction, only five of which, however, were in his opinion of practical importance. He concluded in his Arris and Gale lectures that the right duodenal hernias originated in the fossa of Waldeyer and the left duodenal hernias in the fossa of Landzert. Moynihan's[5] lectures were on "retroperitoneal hernia" but not all duodenal hernias are retroperitoneal. Brown[6], Sir Astley Cooper[7] and Paul and Hill[8] described intraperitoneal duodenal hernias. Moynihan[5] believed that the above fossae were formed by the failure of union of the root of the mesentery with the posterior abdominal wall. He believed, further, that the final size of the hernia depended upon the looseness of the retroperitoneal tissues and the extensibility of the peritoneum. Observers since have generally accepted the view that these hernias were postnatal in origin despite the fact that such hernias have been described by Vogt[9] in a new born baby, by Broesike[10] in a 14 day old baby and by Treitz[2] in a 2 month old child.

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Andrews[11] questioned the generally accepted theory of origin and felt that these hernias were caused by a "congenital anomaly in the development of the peritoneum." He stated that while the small intestine develops rapidly from the single original loop, the colon is more retarded. Growth of the colon across the abdomen from left to right causes its mesentery to form the transverse mesocolon. The cecum grows even more slowly and does not move down into the right iliac fossa until the hepatic flexure has become established. When the final position is taken the mesentery becomes attached to and fused to the posterior abdominal wall. If during this process the small intestine is caught beneath the mesentery of the forward moving colon and is retained by the fusion of the colon to the posterior wall, a right paraduodenal hernia is formed where the superior mesenteric artery and the ileocolic artery would lie in the superior margin of the neck of the sac. Andrews[11] explains the formation of a left paraduodenal hernia in a similar fashion, except that in this instance the small intestine is caught beneath the mesentery of the descending colon. The anterior margin of the sac in this case carries the inferior mesenteric vein and the left colic artery.

Papez[12] believes that both right and left paraduodenal hernias and their surrounding sacs have a more natural origin in the very early embryonic umbilical hernia. He shows that when the small intestines are drawn into the abdominal cavity, they also draw in with them their surrounding peritoneal umbilical sac due to a premature adhesion of its orifice to the duodenal coils and to the mesenteric root. In case the large intestine has been fully rotated to the right side across the duodenum and mesenteric root before the adhesion of the orifice of the sac and retraction of the gut has taken place, it would produce left paraduodenal hernia. In this, the more common form, the abnormal sac is situated to the left of the ascending colon and is suspended from the transverse colon and its mesentery. In case the large intestine has not completed its rotation before the adhesion of the orifice of the sac and retraction of the gut had taken place, the condition would be a right paraduodenal hernia in which the abnormal sac is situated to the right of the ascending colon.

Report of Case

A 17 year old colored girl, was admitted with retained placenta following delivery by a midwife. She died of sepsis twenty days later.

Necropsy revealed an acute endometritis and metritis, with intravascular infection, pyemic abscesses of lungs, infected infarcts of spleen, right ureteritis and pyelonephritis and an internal hernia.

The peritoneal surfaces were smooth, gray and glistening. No free fluid or exudate were present. The intestines were all moderately distended. To the left of the vertebral column, below the transverse mesocolon a hernial sac was found, in the region of the duodenojejunal fossa whose opening pointed to the

View showing the paraduodenal hernia from which most of the small intestine content had been removed. This lies below the transverse mesocolon in the region of the duodenojejunal fossa.

right and measured 6.5 cm. by 4 cm. and its diameter was 10 cm. Along the free border of the mouth of the hernia ran the inferior mesenteric vein and the ascending branch of the left colic artery. The sac contained the terminal portion of duodenum and 35 cm. of jejunum. The loops of bowel were easily removed and showed no evidence of strangulation. No other abnormalities were present.

SUMMARY

A left paraduodenal hernia has its sac opening to the right. The anterior free margin of the sac contains the inferior mesenteric vein and ascending branch of the left colic artery. Callander[13] and Longacre[14] have demonstrated that the mesentery beneath the above named vessels are very loose in the developing fetus. The vessels however offer considerable resistance and therefore the invagination is always beneath them. The descending colon lies to the outer side of the hernia although it may be displaced medially. The amount of small intestine within these hernias may vary from a few inches to its entire length. We have found no record of omentum within the sac.

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THE ATYPICAL SIGNS AND SYMPTOMS IN PERFORATED PEPTIC ULCER

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ACCORDING to reports from leading clinics, about 70 to 80 per cent of the perforated ulcers found at operation are diagnosed before the operation. The remaining 20 to 30 per cent come to the operating table with some other diagnosis. Usually in these cases, the surgeon has been able to establish only the presence of an acute condition of the abdomen, but has wisely opened the abdomen, there to find a perforation in the stomach or duodenum. Fortunately only a few finally betray their true nature on the autopsy table. About 5 per cent recover without benefit of operation. The failure of the surgeon to recognize this rather high percentage of perforated ulcers is no fault of his own. It results rather from a peculiar tendency of the perforated ulcer to manifest bizarre signs and symptoms which cause some patients to succumb without benefit of surgery, and others to carry two abdominal scars, one a McBurney incision for what seemed a typical attack of acute appendicitis, and the other a high right rectus for the perforated ulcer which was present.

The purpose of this paper is to analyze certain signs and symptoms which are frequently distorted in the clinical picture, and to emphasize one or two which seem to be almost constantly present regardless of the other findings.

The classic signs and symptoms and x-ray findings, with which everyone is familiar and which make the diagnosis almost unmistakable, are, first and most important, sudden sharp, knifelike pain in the epigastrium radiating to the entire abdomen. This is accompanied by diffuse, boardlike rigidity, vomiting in more than half the cases, bilateral rectal tenderness, and severe prostration. True circulatory collapse rarely occurs. Although the patient appears cold, clammy, and extremely weak, the blood pressure usually approaches normal. In an analysis of 43 cases by Garver[1], only two had a blood pressure below 100. With careful questioning, about 80 per cent will be found to have had previous symptoms of ulcer. In some the liver dulness is obliterated, but this is not of great diagnostic value unless accompanied by the other findings. The demonstration of a pneumoperitoneum by x-ray or fluoroscope is pathognomonic of a ruptured hollow viscus.

It is well known that many of the perforated ulcers do not conform to the picture shown above. In some, the pain and rigidity is confined to a certain portion of the abdomen. In some there is no

prostration, at times even, a patient may be able to continue work. Some give no previous history of ulcer, and some show no evidence of pneumoperitoneum.

Factors Influencing Severity, Radiation, and Character of Pain

1. Character of fluid
2. Volume of spillage
3. Position of ulcer
4. Sensitivity of peritoneum
5. Presence of adhesions or other inflammatory reaction
6. Gravitation of fluid
7. Elapsed time since perforation
8. Spontaneous closure of opening, "forme fruste"

The outline suggests a few of the variable factors concerned which may influence these signs and symptoms. Since the initial pain is due to a chemical peritonitis, it is probable that a highly acid fluid will cause more pain than fluids containing antacids or bland foods. The most intense pain is produced in perforations following ingestion of alcohol, and the peritonitis which comes after the operation is usually severe in type. One patient observed in Grady Hospital in Atlanta had eaten large quantities of pecans just before perforation. His pain was agonizing, and although the operation was performed early and the abdomen carefully aspirated, he died from a diffuse peritonitis, pieces of pecan being found at autopsy.

The volume of spillage determines the extent of soiling of the peritoneum. Most perforations occur when the stomach is full, and according to Corlette (cited by Garver[1]), are produced by increased peristaltic activity of the stomach. It follows that in most cases of ruptured ulcer, there is a large volume of spillage with gross contamination of the entire peritoneal cavity and consequent diffuse severe pain and rigidity. Those cases in which only a small quantity of fluid is released and only a portion of the peritoneum soiled, naturally will show signs limited to that area.

The position of the ulcer primarily influences the character and radiation of the pain. The percentage of ulcers occurring on the anterior wall of the stomach or duodenum, near the pylorus, corresponds roughly with the number of cases showing the textbook signs and symptoms. From this position, the anatomy of the stomach, duodenum, omentum, and related structures not only guide the fluid over the abdomen, but actually force it closer to the sensitive anterior abdominal wall. Ulcers located on the anterior superior surface of the duodenum frequently direct the fluid toward the right

gutter. Those on the posterior wall of the stomach rupture into the lesser sac where the only access to the general peritoneal cavity is through the foramen of Winslow. Cases of this type usually have colicky pain simulating acute intestinal obstruction. Ulcers on the greater curvature are likely to have symptoms confined to the left quadrants. Toland and Thompson[2] showed that perforated gastrojejunal ulcers following gastroenterostomy typically caused pain and rigidity confined to the left abdomen. This is also true of ulcers on the lesser curvature. Although the radiation of the pain varies with different locations, the first contact of the irritating gastric fluid with the sensitive peritoneum is accompanied by sudden sharp epigastric pain. Subsequently the pain may subside or be directed in numerous directions, but its initial appearance in a sudden unexpected manner is almost invariably present. This constitutes probably the most constant sign associated with perforation, and when it is present one should exercise extreme care in the establishment of the diagnosis before placing the patient on a medical regime.

The sensitivity of the peritoneum has been included merely to mention the individual reaction which different people show toward any type of pain. Some make little display when they are in excruciating pain, while others react violently to little pain. Pre-existing diseases of the peritoneum may also affect its sensitivity.

The presence of adhesions or other inflammatory reaction around the ulcer influences the spread of the fluid. An ulcer rupturing into the region of an inflamed gallbladder and common duct may be confined to the immediate vicinity of the duodenum by adhesions and have little chance to spread to the general peritoneal cavity. The symptoms will be confined strictly to the epigastrium. The similarity to acute cholecystitis is responsible for that diagnosis being made in some cases.

The tendency of the fluid to gravitate in definite directions producing tenderness of the area it traverses, frequently confuses, and accounts for occasional ruptured ulcers being attacked through low abdominal incisions. As previously noted, ulcers on the anterior superior surface of the duodenum discharge their fluid into the general peritoneal cavity above the transverse mesocolon. It may then be guided by the mesocolon down the right gutter to the right lower quadrant where it gives rise to point tenderness and rigidity of the lower right rectus muscle. This tenderness and rigidity frequently exceeds that present in the epigastrium to such an extent that the latter is overlooked entirely, a diagnosis of appendicitis being made and apparently substantiated by an elevated leukocyte count. Careful questioning in these cases will nearly always reveal that the onset was sudden and sharp in the epigastrium, the pain

gradually spreading down the right abdomen to the right lower quadrant. From the right lower quadrant the fluid then gravitates to the pelvis where it is equally distributed on both sides.

Ulcers on the lesser and greater curvature often gravitate down the left abdomen to the pelvis. Because of the omentum, the fluid does not concentrate in the lateral gutter so easily; hence symptoms confined to the left abdomen are not as common as those on the right. Note that regardless of the region of the abdomen which the fluid traverses it eventually descends to the pelvis where it is equally distributed to both sides. This brings to our attention another very important symptom, bilateral rectal tenderness. Sharp tenderness over all the pelvic peritoneum felt by rectal examination is constantly present in all cases of perforated peptic ulcer in which a sufficient quantity of fluid is discharged at the time of perforation to reach the pelvis.

Patients seen immediately after perforation exhibit usually the typical findings. In the course of several hours the initial shock to the peritoneum begins to wear off and the pain subsides. This is called by Kruse[3], the reaction stage of acute perforation. In it a patient who an hour before was in extreme distress now seems to be fairly comfortable; peristalsis may be present, and he may show every sign of recovering from what seems to be "acute indigestion." Sometimes the signs may become so quiescent that the surgeon must rely on the history alone. But here, too, the sudden sharp onset and the presence of rectal tenderness are of value in establishing the diagnosis.

"Forme Fruste" Perforations

There is another type of perforation first described by Singer and Vaughan[4] in 1930, who recognized perforations which run a milder course than acute ruptures, due to the spontaneous closing of the opening. Now generally known as subacute perforations, Singer and Vaughan affixed the decorative title of "formes frustes" perforations, a French expression meaning "atypical forms." The following outline gives the cardinal signs:

1. Sudden onset pain in epigastrium, milder
2. Milder rigidity, localized
3. Signs and symptoms recede quickly, patient appearing much better at end of 24 hours
4. Tendency to develop localized abscess
5. Findings at operation
   a. Small opening
   b. Small volume of spillage
   c. Usually omental plug over perforation

Note that practically all subside in twenty-four hours due to plugging of the small opening by omentum. The percentage of perforations of this type and perforations of chronic ulcer into a surrounding limiting inflammatory exudate of long standing is probably much higher than any of us realize although Deaver drastically stated 15 to 20 per cent of all duodenal ulcers eventually perforate. Some estimate even 25 per cent. Of this high percentage only about 3 per cent take the form of typical acute perforations.

Report of Cases

Case 1. A negro man, aged 30, was admitted to the Surgical Service of Grady Hospital in Atlanta complaining of acute pain in the right lower quadrant. He had been well until about 4 hours before admission when he was stricken suddenly with a sharp but not severe pain in the epigastrium. The

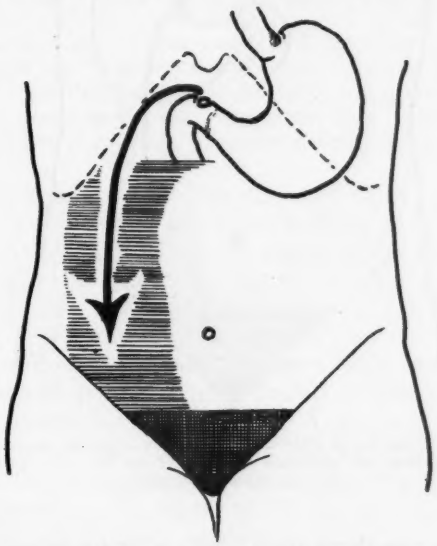

Fig. 1. Case 1. Simulating acute appendicitis. No previous ulcer history. Sudden onset pain in right lower quadrant. Milder pain in epigastrium.

pain spread gradually to the right lower quadrant where it became most intense. The epigastric pain persisted but was not as severe as the lower abdominal discomfort. Examination revealed marked tenderness over the appendix and rigidity over the lower right rectus muscle. There was also some tenderness in the epigastrium and some rigidity of the upper right rectus. Although a diagnosis of acute appendicitis was made, the possibility of an ulcer was considered; therefore, the abdomen was opened through a small right rectus incision opposite the umbilicus. On incising the peritoneum, the characteristic tenacious, cloudy fluid was encountered. The appendix was then examined and found to be normal. The incision was then extended upward.

A perforated duodenal ulcer was found in the location shown. The ulcer was closed with a figure of 8 stitch, and the ulcer bearing area inverted with one layer of Lembert stitches. The abdomen was then thoroughly aspirated. The peritoneum was closed with a continuous chromic catgut suture, and the skin, muscle, and fascia, with interrupted silkworm gut. No drains were used. The patient made an uneventful recovery.

CASE 2. A white man, aged 42, was admitted to the Bulloch County Hospital, Statesboro, Ga., complaining of pain in abdomen. While carrying a load of brick six days before admission, he was stricken with a sudden sharp pain in the epigastrium. He sat down, rested a few minutes, and the pain gradually subsided. He was then able to work the remainder of the day, but his epigastric distress did not completely cease. That night the pain returned, and he began

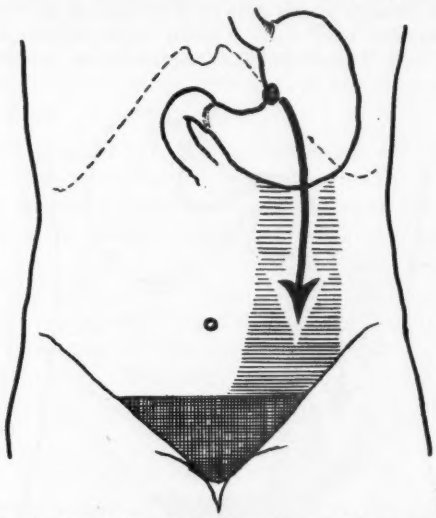

Fig. 2. Case 2. Simulating intestinal obstruction. Sudden onset pain in epigastrium radiating down left abdomen. Pain recurrent and colicky in nature. Peritonitis at time of examination.

to vomit. At this time the pain was colicky in nature and seemed to be worse in the left abdomen, although he was quite sore all over. He did not sleep that night, but the next morning he felt much better and was in little pain. Later in the day, however, the cramping pains returned and were accompanied by intensive vomiting. He also had fever and marked distention of his abdomen. During the three days before admission, he remained in bed and continued to have the colicky pains with occasional vomiting but insisted that he felt better than the first three days, until the day of admission when all symptoms became much worse.

His past history showed indigestion and heartburn over a period of four or five years. He had lost 30 pounds weight gradually over a period of 15 years. No other serious illnesses.

EXAMINATION: Temperature 100°, pulse 80, respiration 20, blood pressure 100/82. General malnutrition. The abdomen was distended. There was mod-

erate generalized tenderness distinctly marked in the left quadrants where rigidity was also present. No masses were felt. No rebound tenderness was elicited. An occasional peristaltic tinkle was heard. On rectal examination, there was diffuse tenderness but no mass.

Red count 4,490,000; hemoglobin 80 per cent (Tallqvist); white count 6,300, with 64 per cent polymorphonuclears. Urine contained a trace of albumin, trace of sugar, 3 to 4 pus cells, and occasional red blood cell and granular cast. X-ray of abdomen: There was no evidence of pneumoperitoneum. Multiple fluid levels were seen. A diagnosis of intestinal obstruction, etiology undetermined, was made.

Under 150 mg. procaine hydrochloride crystals spinal anesthesia, a small right rectus incision was made opposite the umbilicus. The peritoneum was indurated and red. Cloudy yellow tenacious fluid with flecks of exudate was present. Diffuse peritonitis had already developed. The incision was then enlarged upward, and the stomach and duodenum examined. A large indurated mass measuring about 4 by 3 by 2 cm. was found midway on the lesser curvature. In the center of this was a small perforation. This was closed with a figure of 8 suture and inverted with a layer of Lembert sutures. The abdomen was then aspirated and the peritoneum closed with continuous chromic catgut. Because of the friability of the tissue, the fascia was included in the suture. The remaining layers were closed with interrupted silkworm gut closely placed. No drains were inserted. The patient died three days later.

The foregoing has been an attempt to bring before you some of the variable factors which affect the findings in perforation. The conditions most often confused are appendicitis, cholecystitis, and intestinal obstruction. It is interesting to note that most of the cases in which the patient had colicky pain simulating intestinal obstruction, that the perforated ulcer was found on the lesser curvature, or posterior wall of the stomach. In two cases reported by Coleman[5], one showed typical pain of intestinal obstruction and this diagnosis was made before operation. A perforation on the posterior wall of the stomach discharging into the lesser sac was found. In the other, the same preoperative diagnosis was made. In this case, the perforation was found on the anterior surface near the lesser curvature. Both cases recovered.

Rivers[6], in an analysis of peptic ulcer pain, states:

> When the pain of gastric ulcer shifts definitely to the left, slightly upward or to the back; when the pain of a duodenal ulcer radiates toward the right, upward over the area of the liver or through to the back, one can correctly assume deep penetration or partial perforation of the lesion.

This observation is of value in determining when a peptic ulcer under treatment threatens acute perforation.

There is little doubt many more perforations occur than are recognized. That accurate diagnosis of these lesions will save many lives is doubtful, because fortunately the majority of the conditions

which confuse the picture are themselves acute surgical emergencies. However, with the growing popularity, in operations on the upper abdomen, of the subcostal or transverse incision, which is comparable in strength of union to the McBurney incision, it is important that an accurate diagnosis be made, especially since an incision of this type is inadequate for thorough exploration of the abdomen.

Summary

1. Seventy to eighty per cent of perforated ulcers are diagnosed correctly before operation, 20 to 30 per cent have other diagnoses, usually acute appendicitis, intestinal obstruction, or cholecystitis.

2. The cardinal signs and symptoms of acute perforated ulcer were reviewed.

3. Factors influencing severity, radiation, and character of pain in perforated ulcer were listed.

4. Signs and symptoms of "forme fruste" perforations were given.

5. A case of ruptured ulcer with gravitation of the fluid down the right gutter where it simulated appendicitis was reported.

6. A case of ruptured ulcer located on the lesser curvature with colicky pain simulating intestinal obstruction and radiation down the left abdomen was reported.

7. One symptom and one sign were stressed: (a) Onset of sudden sharp pain in the epigastrium. (b) Bilateral tenderness on rectal examination. In practically all the atypical cases these two were present.

8. The use of the subcostal incision was mentioned.

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MASSIVE DILATATION OF THE COMMON BILE DUCT

Presentation of a Case With Report of Autopsy

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ALTHOUGH the literature on dilatation of the common bile duct is comprehensive and readily available, a correct preoperative diagnosis is made almost as rarely as the phenomenon itself occurs. The case here presented, supplemented by a report of the postmortem examination, illustrates the salient features of this unusual condition and is recorded to assist in making it better known..

A review of the literature by McWhorter[1] in 1924 included 49 cases. He observed that in none of these cases was the correct diagnosis made before operation, that failure to recognize this type of obstruction and to provide internal drainage had resulted in an operative mortality of 71 per cent, and that all cases in which an operation had not been performed had resulted fatally. In more than 50 per cent of these cases the symptoms had developed before the patient was 10 years of age, he noted. Five years later, Stoney[2] placed the number of authentic cases at 67 and reported the average age of the patients as 15½ years with a history of symptoms lasting for from 2 months to 36 years. Willis[3], giving a resumé of 58 cases, observed that 50 per cent of the patients were under 15 years of age and that only 10 per cent were more than 25 years old.

Recently, Berkley[4] added the report of a case to the 100 cases, including his two, that Gross[5] found recorded in the literature, and he noted that of the 53 cases occurring in childhood or presenting symptoms from childhood to adult life, only 3 had been correctly diagnosed preoperatively. As the syndrome becomes more and more familiar, doubtless the percentage of diagnostic error will be steadily lowered for the clinical symptoms and the physical findings are fairly characteristic. Failure to recognize the true nature of the condition even at operation has in the past been an important factor in the high mortality.

The usual symptoms are recurrent attacks of jaundice, pain in the upper portion of the abdomen and a palpable tumor occurring especially during childhood or early adolescence. Jaundice, the most constant symptom, is not always present. According to Gross,[5] the condition occurs three times as often in females as in males.

Most authors regard cystic dilatation of the common bile duct

as a congenital anomaly although others describe it as idiopathic or acquired. The etiology, however, remains obscure.

From the standpoint of treatment, on physiologic and theoretical grounds, as Zinninger and Cash[6] observed, primary anastomosis to establish as early as possible an adequate communication between the biliary tract and the intestine is the most desirable procedure. As a measure against infection, excision or partial excision of the cyst, when possible, reduces stasis to a minimum. These procedures have been more effective than treatment by drainage alone, as formerly. In the case here described an anastomosis was made between the greatly dilated common bile duct and the duodenum.

Report of Case

A. T., a white girl, aged 2 years, was sent to Riverside Hospital on April 6, 1939, on account of jaundice of three months' duration. She was the only child of healthy parents of Greek extraction, who had been married for four years. Normal at birth, except that she weighed only 5 pounds, she had gained 10 pounds the first year, the history revealed, and she had contracted none of the infectious diseases of childhood. The mother had observed no abnormalities in the child other than peculiar tastes as to food, in particular an excessive desire for sugar, and she had allowed her to drink several Coca-Colas daily.

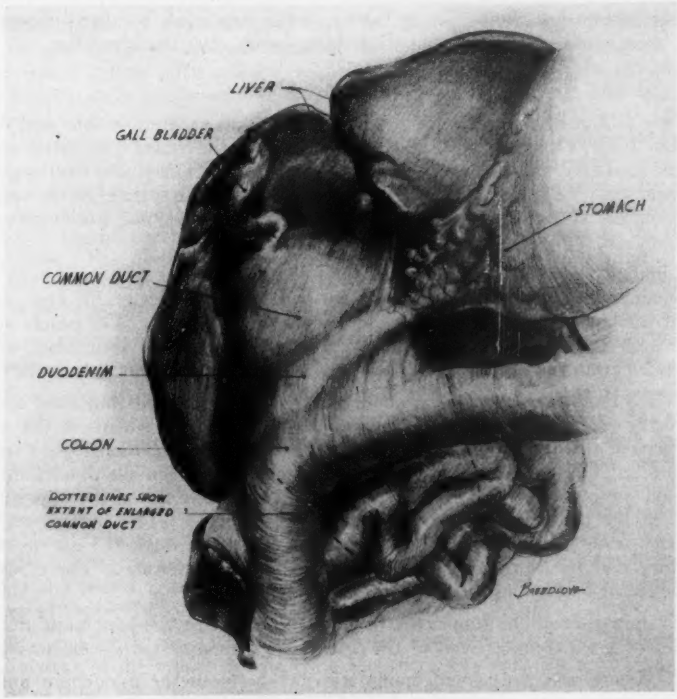
In the summer of 1938, when the patient had suffered an attack, believed to be an infection of the respiratory tract, the mother had noticed that her skin was slightly yellow for a time; this yellowish hue had reappeared in January, 1939, and had continued to deepen up to the time of admission, with increasing malaise and occasional attacks of vomiting accompanying this change. Although the mother had noted no other gastrointestinal irregularities, she had observed that the stools had been pale and whitish in color most of the time since January, although at intervals they had appeared to be greenish, and that they had been white continually for the three weeks immediately preceding admission. She had noticed no change in the color of the urine.

On entry, the patient weighed 25 pounds. Her skin, hair and eyes were very dark. The color of the skin and the sclera gave abundant evidence of the presence of jaundice. The abdomen was symmetrical, but generally distended. On palpation, a soft sharp-edged resistance was felt, extending about 2 inches below the costal border, in which a notch was discernible at about the position of the gallbladder. This swelling, soft, homogeneous and not tender, was apparently the liver. The spleen could not be felt. From under the right lobe of the liver extending downward below the level of the umbilicus a mass apparently 2½ or 3 inches wide could be palpated. It seemed longitudinal rather than transverse in shape, was not tender, and was fairly well fixed to the deep structures. Either on top or as a part of it, several nodules about ½ to ¼ inch in diameter and not especially tender could be felt. On bimanual palpation this mass would transmit the pressure from the hand on the abdomen to the one placed over the lumbar region on the right side, but it was not the lumbar swelling characteristic of a tumor of the kidney.

Urinalysis showed the urine was clear, amber in color and alkaline; the specific gravity was 1.005, there was a slight trace of albumin but no evidence of sugar, and the test for the presence of bile was positive. Examination of

the blood revealed hemoglobin 70 per cent, erythrocytes 4,200,000, leukocytes 14,600, polymorphonuclears 30, lymphocytes 64 and eosinophils 6. The bleeding time was from 10 to 12 minutes and the coagulation time 12 minutes; the platelet count was normal. The icterus index was 100. The Kahn test gave negative results.

Roentgen studies demonstrated a shadow, apparently of soft tissue, continuous with the liver shadow, extending downward from the abdomen into the upper portion of the pelvis slightly more on the right side than on the left. The kidneys were not well outlined. After the injection of 10 c.c. intravenously, diodrast solution appeared within 5 minutes in both kidneys, showing



Artist's drawing from the autopsy material.

them to be normally placed with the pelves and calices apparently normal. When the solution had collected in the bladder, its usual round symmetrical shape was altered by a cut-off square margin on the superior surface, apparently caused by a mass lying against it that was a part of the liver or hepatic system.

Preparatory to an exploratory operation, the patient was put on a diet high in carbohydrates and was given "klotogen" tablets and bile salts, one tablet three times a day. After six days the bleeding time was 4½ minutes and the clotting time 6 minutes. The hemoglobin had increased to 78 per cent and the erythrocyte count to 4,520,000; the leukocyte count was 11,600 and the differential count showed polymorphonuclears 44, lymphocytes 54 and eosinophils 2.

At operation, on April 20, the liver was but slightly enlarged, the gallbladder was small and the stomach was normal. A large fluid-containing tumor extended up under the liver and thrust forward the first portion of the duodenum. It was about 3 inches in diameter and 5 inches long; when aspirated, it contained green bile. After the duodenum had been freed for a short distance, the tumor was incised, and a large quantity of green bile escaped, 400 c.c. of which was recovered. The swelling was then opened, and an attempt was made to locate the ampulla of Vater from within. It was, however, not possible to find any opening which might be that of the common bile duct. An anastomosis was made between the first portion of the duodenum and the swelling, which was certainly the common bile duct.

The patient left the operating table in a fair condition, but her temperature and pulse rate rose rapidly. She died thirty hours after the operation.

Autopsy

Two and one-half hours after the patient's death an autopsy was performed by Dr. L. Y. Dyrenforth. The anatomic diagnosis included congenital atresia of the common bile duct with massive dilatation and bile stasis, icterus gravis, chronic passive hyperemia of the liver, congestion of the spleen, recent surgical anastomosis of the duodenum and the common bile duct, and pulmonary congestion.

The body was that of a white girl of the dark-haired Mediterranean type with abundant hair, well developed but moderately underweight and undersized for her age. The skin and sclera were deeply icteric. The pupils of the eyes were equal, regular and contracted. A midline recent abdominal wound extended from the umbilicus to the symphysis pubis, with stay sutures in place.

When the body was opened, about 200 c.c. of bile-tinged thin fluid escaped from the peritoneal cavity. The peritoneal layers and the omentum, the serous surfaces of the organs and the tissues of the walls of the abdomen were stained with bile. The suture line of a recent anastomosis between the duodenum just beyond the pylorus and a bladder-like tumor completely filling the upper quadrant of the abdomen on the right side, was observed. The tumor was continuous with the gallbladder, into which it opened through the ductus chole dochus, and its lining was a smooth, deep green membrane. There was no evidence of leakage in the anastomosis.

Examination of the lumen of the duodenum and of the pancreatic duct, as well as the entire inner wall of the tumor sac, demonstrated the following:

1. There was no normal communication between the duodenum and the cystic enlargement which was taken to be the dilated common bile duct.

2. A clearly defined papilla of Vater was present, opening into the duodenum about 2.5 cm. below the pyloric ring posteriorly, but the duct following along this opening entered the main pancreatic duct. No communication with the extrahepatic bile ducts was found.

3. The hepatic duct opened into the sac just beyond the neck of the gallbladder. This organ was dilated and thickened and contained a mucoid secretion tinged with bile. The communications of the hepatic duct in the liver were patent so far as dissection of the larger branches showed.

The liver was enlarged and extended downward for a distance of nearly 3 cm. below the right costal border and the ensiform cartilage of the sternum.

In the thorax pulmonary congestion was evidenced by the presence of some 50 c.c. of clear yellowish fluid within the pleural cavities. The lungs were

crepitant in their upper lobes, but patchy red areas were present that exuded quantities of frothy, blood-tinged serum on pressure. No adhesions between the pleural layers were observed, and the visceral pleurae were smooth and of a dull reddish color. No hilus glands were prominent, and normal involution of the thymus gland was evident.

Except for the presence of a demonstrable quantity of clear serum in the pericardial sac, the heart and pericardium showed very few changes. The left ventricle of the heart was slightly hypertrophic. Dissection of the organ revealed approximately normal valvular openings and a smooth grayish endocardium. The papillary muscles were not grossly affected. The diaphragm extended to the fourth intercostal space on both sides.

The contents of the body cavities were removed en bloc to facilitate dissection and studies in reconstruction of the abnormal extrahepatic ducts. The liver was firm and putty-colored with blotchy areas of tan. The gallbladder had a thick wall, contained bile and was connected by a cystic duct with the dilated common duct. The esophagus, stomach and intestinal tract offered no abnormalities. The spleen was slightly enlarged and was soft with prominent corpuscular markings in the dark purple pulp. The adrenals were partially softened. The kidneys were of average size and appearance for their age, showing a moderate but persistent fetal lobulated surface; the ratio of cortex to medulla seemed average, and there was nothing of note in the pelves or calices. The ureters, the urinary bladder and the external genitalia were normal in appearance. The reproductive organs also appeared to be normal. There were no enlarged mesenteric lymph nodes.

The drawing from the pathologic specimen demonstrates the significant findings.

Microscopic examination showed the liver cells to be fairly well preserved. The only prominent change was demonstrated with fat stains, sudan III showing the presence of fat, in the form of fine droplets, within the cell borders diffusely. Much pigment was also present in the cells and within the cytoplasm of the phagocytes and Kupffer's cells of the sinusoids. The capillaries were congested with erythrocytes. There were foci of lymphoid cell accumulations resembling miliary abscess. A peculiar fibrosis occurred beneath Glisson's capsule, and there was present a pronounced and uniform fibrosis involving mostly the portal triads. They in some instances showed extensive accumulations of connective tissue.

Microscopically, the outstanding condition observed in the gallbladder was hypertrophy of the muscle in the wall, but there was very little fibrosis. The vesicle mucosa was atrophic, and the pigment was stained.

The histologic sections from various parts of the wall of the tumor sac or dilated common bile duct all showed the absence of a secreting membrane. The wall was a structure of rather thick connective tissue containing some evidence of smooth muscle fibers and elastica, collagen and areolar connective tissue. The inner border suggested the intimal layer of the large blood vessels and, so far as could be determined, was devoid of a secreting membrane.

Sagittal sections through the altered papilla of Vater showed an ordinary papillary lining composed of high columnar epithelium. A modified serial section technic failed to disclose the presence of any vestigial side opening along the distal few millimeters of the papilla of Vater or the pancreatic duct. The entrance of the duct into the duodenal mucous layer was demonstrable but not noteworthy. The surface of the mucous membrane was covered with a thin layer of purulent mucoid material.

Examination of the other organs revealed no further contributory evidence. Follicles were prominent in the spleen, which was hyperemic, and its pulp was filled with destroyed pigment. The lungs contained alveoli filled with serum. Neither the myocardium nor the kidneys gave evidence of significant changes. The adrenals were too soft to stain well.

Conclusion

Even a careful search at autopsy failed to reveal an opening from the liver into the duodenum in this case. A duct leading from the pancreas to the duodenum was discovered, but it was empty where it was expected the ampullar body would be located. If there was no opening at the time the child died, the question arises as to whether there ever had been one. If not, it is certainly a matter of remark that persistent and increasing jaundice was not outstanding in the patient's history. In some of the cases reported there was no mention of the presence of jaundice, and in others it recurred intermittently. In the case here presented it is difficult to understand how dilatation of the duct might have caused it to fold over on itself in such a manner as to block the opening.

Another question of interest is whether the liver could stand for almost two years the back pressure incident to the obstruction of the common bile duct, as appeared to be true in this case. Yet the liver showed no gross evidence of insult, and the sections examined microscopically demonstrated the presence of only mild disturbances.

It is hardly to be expected that a bile duct, dilated so excessively as in this case, could be present through any considerable period of life without causing pronounced constitutional symptoms. That preoperative diagnosis has been incorrect in almost 100 per cent of the cases reported is therefore understandable.

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Riverside Hospital.

A SIMPLIFIED AUTOMATIC INTERMITTENT IRRIGATOR

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RECOGNIZING the need for a simplified automatic irrigator to use in urology and other branches of surgery, preliminary investigation was started three years ago to design such an apparatus. At first it was thought that a secondary reservoir could be mounted on one end of the beam of a simple balance, with a rotary valve as the fulcrum. The addition of water into the reservoir was to produce an increase in the force acting on that end of the balance and rotation of the beam, thus turning the valve. With the reservoir end of the beam balanced upward, water would flow into the intermediate reservoir; with the reservoir end of the beam down, water would flow out of the reservoir and into the bladder. A mercury counterweight, by flowing from one end of the beam to the other, would change the resultant force enough to overcome equilibrium, and then hold the secondary reservoir in the correct position for filling or emptying before the beam was overbalanced to the opposite position.

Two instruments using these principles were successfully designed but neither was very efficient due to friction of the valves and leakage. These faults were not overcome. After experimenting with various types of metal and rubber valves, it was decided to abandon the use of valves entirely and employ a siphonage system to empty the secondary reservoir.

The drawing of the apparatus which is used at present shows the general design. A secondary reservoir rests on one end of a beam balance and is 18 inches above the bed. Water from a primary reservoir drips into a secondary reservoir at any desired speed. When the intermediate reservoir has filled to within 50 c.c. of capacity, it overbalances the fixed and movable counterweights on the opposite end of the beam. The weight of the filled reservoir comes to rest on the rubber drainage tube, constricts it at this point, thus preventing drainage from the bladder. The secondary reservoir continues to fill and when completely full empties by siphonage into the bladder. When the secondary reservoir is almost empty, the fixed counterweight overbalances it, thus removing the constriction of the drainage tube allowing the bladder to empty. The mercury counterweight flows to the distal end of the tube adding enough weight to keep the reservoir overbalanced until it has been again filled to within 50 c.c. of capacity.

It will be seen that the apparatus delivers a definite amount of fluid for each irrigation, and thus differs from the types of urologic irrigating equipment which fill the bladder to a certain pressure. The amount of fluid which is to be used with each irrigation is predetermined by measuring the capacity of the bladder before the automatic irrigator is used. The fixed and movable counterweights are varied so that the requisite amount of fluid will be dispensed for each irrigation.

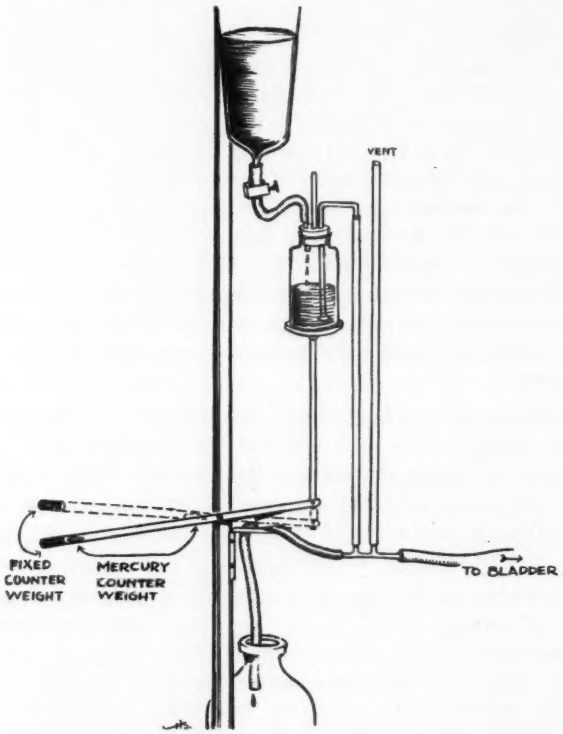

Diagram of the apparatus.

With this apparatus the bladder empties spontaneously and siphonage is not possible, since a vent breaks the suction which would result if the fluid ran through a closed tube into the drainage bottle.

In the construction of such an instrument the following principles are followed:

1. All moving parts are made as frictionless as possible.

2. A fixed counterweight heavy enough to balance the reservoir containing 50 c.c. of water is essential.
3. A sliding counterweight is necessary. When the movable counterweight is at the distal end of the beam it should be heavy enough to balance the amount of water to be dispensed.

The time interval of irrigation is controlled by one factor only, that is, the rate of flow of the fluid into the secondary reservoir.

The instrument is suggested for use following transurethral prostatic resection and in other conditions where intermittent irrigation is necessary or desirable. The first piece of apparatus which was made has now been in use for four months. In one case it irrigated a neurogenic bladder automatically day and night for a period of two months without attention except for the refilling of the primary reservoir twice daily. Before the automatic irrigator was used the patient had an intractable infection. After instituting this type of intermittent irrigation the infection rapidly became insignificant and remained so.

The Southern Surgeon

Published Monthly by
The SOUTHERN SURGEON PUBLISHING COMPANY
701 Hurt Building
ATLANTA

L. MINOR BLACKFORD, M.D.
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Managing Editor

Subscription in the United States, $5.00

VOLUME IX MARCH, 1940 NUMBER 3

"STIMULUS TO THE YOUNG MEN"

Two months ago we published a prophecy as to THE SOUTHERN SURGEON made by the beloved and inspiring Dr. Haggard. One who had the privilege of knowing him better is paying tribute elsewhere to this courtly gentleman but it is appropriate here to repeat his words again:

> It will be a great stimulus to the young men to perfect their work, to record it, and to make additional studies that are so important in the preparation of medical papers and case reports, and are of such paramount value to the man who studies them.

The great surgeon's foresight is being justified. During 1940 twenty-six authors have contributed to these pages: more than half of them, fifteen to be exact, were born during the twentieth century. While four of our contributors this year have passed 50, two have not yet reached 30! That's probably a record of some sort: at least we are proud of the record. The average age would be even younger except that the January SURGEON was devoted largely to papers read before the 1939 Assembly. The February and March numbers have been bare of Assembly papers, the first ones without such. These two issues are not so weighty with names already famous as some we have published, but otherwise they will hardly suffer by comparison.

Dr. Haggard was not the first great man in medicine to emphasize the importance of bringing out the young men. Twenty-eight years earlier Dr. Osler had achieved most distressing notoriety when he was reported as an advocate of chloroforming men when they reached 60. This was suggested facetiously to lighten the solemnity of his formal farewell at the Hopkins. However, in that address, "The Fixed Period," he did emphasize that the greatest discoveries in medicine had been made by those under 40. His interest in bring-

ing out the young men who worked under him was proverbial and he inspired them to great works. Surely he was never more sincere than when he said, "The young men should be encouraged and afforded every possible chance to show what is in them." Further along in the same essay he emphasized that the judgment of maturer years is needed to guide youth.

We would reiterate that THE SOUTHERN SURGEON will never turn down a paper because its author has reached years of discretion. Such men, even though they may not be as fecund in startling innovations, may still have excellent original ideas, and their experience not only tends to keep them from going off half-cocked but also lends the weight of mature judgment to their essays. We shall always welcome their efforts (though we hope to be excused from ever publishing what they have already published elsewhere). Such men, however, should have no need of the stimulus of THE SOUTHERN SURGEON, and the prestige of their names is such that their offerings will be accorded a respectful hearing by any editor. It is the surgeon who is young enough to accept suggestions as to the mode of presenting his ideas, who needs encouragement, who will not be offended if a paper is returned to him, that we particularly wish to have write for this journal. And these monthly appearances this year have proved that they can be had.

THE PREPARATION OF PAPERS

Since we have just emphasized that we want and expect young men to send their efforts to the SURGEON, may we address a few lines to them in confidence:

First, please read over carefully the conditions under which papers are accepted. These conditions have been printed in each issue of this journal: they will be found on the last page of the present number. We sometimes get a very bitter letter from a contributor when he receives the bill for the cost of his illustrations. This page also gives instructions as to the means of preparing your references. You would be surprised to know how often references are not written in the proper form. Sometimes they are actually inaccurate.

A good, snappy case report is always acceptable. It requires some work to prepare such a case report: you can't just copy the original notes from the hospital chart. Remember, original case reports are the building stones of clinical medicine. All editors are partial to short papers, —and so are most readers.

A series of cases is in some ways even better. But please, please be careful about drawing conclusions from your figures. If you toss a coin three times, it may come out heads all three times, but if you toss a coin a million times it will come out heads just about 500,000

times. If you perform a certain operation on two patients and one dies, that does not prove the general mortality from that operation is 50 per cent. If you do the same operation seven times and all seven patients live, that does not prove the operation carries no mortality. Once the writer was working on a series of 37 cases, and he thought his figures proved a point. He showed them to a statistician who went into a huddle with himself and after consulting his table of logarithms announced, "There is one chance in 80 that your figures are of no significance." When we said that seemed pretty conclusive to us, he laughed at us. Raymond Pearl's book "Medical Biometry and Statistics" should be required reading for any one who wished to prove anything in medicine by means of statistics.

Another thing, no surgeon wants to read a rehash of a chapter in some system of surgery: he probably has a copy of the same system. Don't attempt to read every paper ever published on the subject you are interested in. Remember the famous old comedy of Moliere: a man is on trial for stealing sheep, his lawyer begins his defense with Adam and the judge constantly interrupts him, "Let's get back to those sheep." Whatever you do, don't cite a reference you haven't read. It is perfectly permissible of course to say, "According to Jones[1], Smith reported—" If you must write an exhaustive treatise, don't send it to the SURGEON.

Good illustrations add interest and clarity to your paper. They also attract attention. If you use them, each illustration must be numbered, and on the back of it either write your name and the title of your paper with a soft pencil or, better still, typewrite it on a slip of paper and paste that securely on the back. If there is any possible doubt as to the top of the picture, indicate the top. Photographs should be printed on glossy paper, and if an x-ray is to be used you should send a glossy print of it so made that the black of the original plate is black in the print. The legends to go under the pictures are not attached to the pictures, but are typed on one separate sheet.

Remember that the Editorial Council is human too. If your paper presents a neat appearance and is double spaced (our contract with the printer requires all copy to be double-spaced) it suggests that the work that went into it was also carefully done. Good spelling helps.

All of these suggestions and many others will be found set forth in more detail in a little book, "Medical Writing," which may be obtained for $1.50 from the headquarters of the American Medical Association.

WILLIAM DAVID HAGGARD

President of The Southeastern Surgical Congress, 1936
September 28, 1872—January 28, 1940

To declare one's inability to find words to express certain thoughts and emotions often is a rank exaggeration, but such is not the case when one attempts to formulate adequate language to describe the keen sense of grief felt in the death of such a man and such a friend as W. D. Haggard. Perhaps the shock of the sudden announcement of his passing tends to enfeeble the pen seeking to write a suitable obituary. We knew that a recent illness of Dr. Haggard had left him with a weak heart, but we had seen and heard him since, and he appeared to possess all his accustomed vigor and good spirits. Now to think of him as dead seems impossible. He always was so much alive. How can we go on without him, without his magnetism, his clear speaking, his wit and humor, and his matchless eloquence? Again it is no flattery to say there is no one to fill his place. There may be those just as brilliant and charming, or those with equal knowledge of medicine or with equal skill with the scalpel, but to find another of superior attainments in so many fields as Will Haggard will require a long time, certainly in the dear country he loved to laud so often, the country we call the South. He was a unique character which comes only once in a generation, or perhaps in several generations, and above all he drew all men towards him. This after all was the greatest thing, we loved him.

Dr. Haggard was a man with multitudes of friends, and among these none admired him and his talents more than two great surgeons who also so recently passed into the great beyond, William and Charles Mayo. It was appropriate therefore that the last writing he ever did, among voluminous articles, was an unfinished memorial to these famous brothers. Although honored as few doctors in the United States ever have been, there was nothing of the egotist about W. D. Haggard. No compliments ever turned his head, but he never missed an opportunity to sing the praises of his confreres and competitors.

To recount the details of his interesting life, reading like a romance, Dr. Haggard was born in Nashville, September 28, 1872, the son of Dr. W. D. Haggard, Sr., and Jane Douglas Haggard. The older Dr. Haggard was one of the best known surgeons of his day, and was the first president of the Southern Surgical and Gynecological Association, in 1888. Today the organization is known as the Southern Surgical Association. He was a pioneer in gynecology, and for many years held the chair of abdominal surgery

in the Medical Department of the University of Tennessee, then located in Nashville.

W. D. Haggard, Jr., was graduated in medicine from the University of Tennessee in 1893 and began practice in Nashville. In 1896 he became assistant professor of gynecology in his Alma Mater, and in 1900 was made professor of gynecology and abdominal surgery. He served in the latter capacity until 1912, when he became professor of surgery and clinical surgery in Vanderbilt University School of Medicine. All these years Dr. Haggard was building a large private practice in surgery and gynecology. He held the positions of surgeon and first president of the staff of St. Thomas Hospital and was a visiting surgeon at Vanderbilt University Hospital. During the World War he served as major and later as lieutenant colonel in the medical corps of the United States Army, acting as surgeon to Evacuation Hospital No. 1 at Toul, France, 1918-1919, and also as consultant in surgery at the Mesves Hospital Center in France. He acted for a time as a member of the Advisory Board of the Division of Surgery in the Surgeon General's office.

Dr. Haggard was intensely interested in medical organization, and devoted much time and effort to the many important societies and associations of which he was a member. His activity, wise counsels and popularity eventually made him president of most of the medical and other organizations to which he belonged. He was a member of the Nashville Academy of Medicine (president 1902), and the Tennessee State Medical Association and Middle Tennessee Medical Association, serving also at one time as president of these societies. He was one of the live wires of the Southern Surgical Association, serving as secretary from 1903 to 1916, and president in 1917. In the American Medical Association he held many positions of importance, being a member of the House of Delegates in 1905 and 1906 and again in 1922. He was secretary of the section on surgery in 1898-1899, and 1909-1910, and chairman of the section in 1916-1917. He also served continuously as a member of the Council on Medical Education and Hospitals from 1912 to 1921. In 1925-1926 Dr. Haggard was president of the American Medical Association.

The University of the South conferred upon him the degree of Doctor of Civil Law in 1931. He was instrumental in aiding the formation of the American College of Surgeons in 1913, and served that body for many years as a member of the Board of Regents, becoming president in 1933. It was in celebration of this election that Dr. Haggard was honored by his fellow practitioners and other friends with a dinner at the Hermitage Club in Nashville, with an

Photograph by Bachrach.

DR. W. D. HAGGARD

President of The Southeastern Surgical Congress, 1936.

overflow audience. Few members of the profession have heard such eulogies as were showered upon W. D. Haggard at this time. He belonged to the Southern Medical Association, American Surgical Association, Pan-American Medical Congress, Sociètè International de Chirurgie, Society of Clinical Surgery, and the Interstate Postgraduate Medical Association of North America, being president of the last in 1929. The final honor of this kind to be conferred upon Dr. Haggard was his choice as president of The Southeastern Surgical Congress in 1936. He always expressed the highest esteem for this young organization and predicted for it a happy, useful career. The Congress appreciated the interest in its affairs taken by so distinguished a surgeon and felt that it was mutual honor to have him serve as president.

Active membership in so many societies necessarily meant that Dr. Haggard must prepare an endless number of surgical papers and other addresses to be presented on various occasions. It would be interesting to note the titles. Most of them dealt with practical discussions of such subjects as appendicitis, goiter and surgery of the stomach and colon. He was such a fluent speaker, and so well informed on the subject matter that he rarely found it necessary to read from manuscript or refer to notes. Many of his papers are included in a volume entitled "The Romance of Medicine and Other Addresses," published in 1927. Other articles appear in a later book entitled "Surgery, Queen of Arts and Other Papers and Addresses," published in 1935.

Dr. Haggard found time to make valuable contributions to his community aside from those of a professional nature. In 1924 he was president of the Exchange Club. During his incumbency it was largely through his influence that one of the principal streets of Nashville was widened. In 1926 he was the recipient of the Kiwanis Cup as being Nashville's outstanding citizen. At one time he headed the Nashville Community Chest. He was a member of Alpha Omega Alpha and Alpha Kappa Kappa, a Democrat, Episcopalian and belonged to the Bellemeade Golf and Country Club and the Hermitage and University Clubs. He had served as president of the last two groups. His tireless energy led him to pursue his hobby of horseback riding, and he won a cup at the Tennessee State Fair in 1927 as the best man rider of the show.

Dr. Haggard was twice married, first in 1898 to Miss Mary Laura Champe of Nashville, who died in 1920. In 1926 he married Miss Lucile Holman, of Nashville. His second wife died in 1932. Survivors are three children, a daughter, Mrs. Burgess Askew, Jr., and two sons, William D. Haggard, Jr., and John Holman Haggard, and a granddaughter, Jean Haggard Askew.

Death came in his sleep January 28, 1940, at the Breakers Hotel, Palm Beach, Florida, where he had gone for a short vacation. In his latter years he rarely went away without taking Billy and John with him, the idols of their father's heart. The funeral, in Nashville, two days later, was attended by one of the largest gatherings ever assembled in the city on a similar sad occasion. Many friends from a distance were present. At the time of this writing plans are being made for a memorial meeting.

Thus passes one who will be missed from our ranks for a long time. He shall be missed in many ways but perhaps in no capacity more than as a presiding officer at a meeting or at a banquet. Here he was indeed in his element. How the audience would hang on his bright sallies and repartee, never biting but always tactful! As a raconteur he had few equals. His command of the English language was unexcelled. Few men of his generation brought to those around the festive board such delight and inspiration as W. D. Haggard. The beauty of all, however, was to know that behind this seeming frivolity lay a serious mind and a heart of gold. You could always depend upon this friend. He was sincere and honest to the core. Truly he did a lot of living in his sixty-seven years. No man ever had more fun or contributed more pleasure to others, nor has one among us worked harder or given more of himself to his profession and to his patients. He leaves a fine example for posterity of happily rounded beneficent manhood.

FRANK K. BOLAND, M. D.

BOOK REVIEWS

The Editors of THE SOUTHERN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The Editors do not, however, agree to review all books that have been submitted without solicitation.*

ENDOCRINE GYNECOLOGY. By E. C. HAMBLEN, B. S., M. D., F. A. C. S., Associate Professor of Obstetrics and Gynecology, Duke University School of Medicine; Gynecologist in Charge of the Endocrine Division and Sex-Endocrine Clinic, Duke University Hospital, Durham, North Carolina. Foreword by J. B. COLLIP, M. D., Gilman Cheney Professor of Biochemistry and Pathological Chemistry, McGill University, Montreal. 453 pages, with 169 illustrations and 4 color plates. Price, $5.50. Springfield and Baltimore: Charles C Thomas, Publisher, 1939.

Sometimes, contrary to the opinion of those who think he is an old grouch with horns, a tail and a cloven hoof, a reviewer gets hold of a book that arouses so much enthusiasm in his breast that he has to restrain himself. This book of Dr. Hamblen's is such a book. With all the folderol and half-baked stuff that passes for endocrinology it is positively refreshing to get hold of a book in this field that is so delightfully conservative. One notes, in passing, that when the author is recording a case that taxes credulity he is careful to say "—— reports," and thus escapes endorsement. We shall hope to see a new edition every three or four years, because in a province where so many advances are being made, new editions will be often necessary.

The first section deals with "Sex-Endocrine Principles." Usefully it lists preparations commercially available that have been found valuable at Duke. At the same time, the author admits that some products not listed may be also valuable. The second part is devoted to "Gynecic Physiology," the third to "Endocrinopathic Gynecology."

This book is recommended with enthusiasm. While those already learned in this field will find it convenient and useful, the book is above all practical and it is therefore especially recommended to the general man.

FUNCTIONAL DISORDERS OF THE FOOT: THEIR DIAGNOSIS AND TREATMENT. By FRANK D. DICKSON, M.D., Orthopedic Surgeon, St. Luke's; Kansas City General; and Wheatley Hospitals, Kansas City, Missouri. Providence Hospital, Kansas City, Kansas, and REX L. DIVELEY, M.D., Orthopedic Surgeon, St. Luke's; Kansas City General; and Wheatley Hospitals, Kansas City, Missouri. Providence Hospital, Kansas City, Kansas. 296 pages, with 202 illustrations. Price, $5. Philadelphia, London and Montreal: J. B. Lippincott Company, 1939.

There is a tendency to think of functional disorders as the manifestation of the innate perversity of certain disagreeable whining females or, less frequently, of certain males for whose existence no excuse can be found. It is something of a novelty then to find that most of the troubles we experience with our feet are due, at least in the earlier stages, to "functional disorders."

The authors of this book begin with a discussion of the evolutionary development of the human foot, its anatomy and physiology and the primary causes of foot imbalance. In the section on the foot of the child, they point out that imbalance is frequent, often resulting in such things as "growing pains" (which parents are only too apt to accept as natural), knock knees and bow legs. They continue with chapters on the foot of the adolescent and of the

adult. In the chapter on shoes one reads that in this country there are about 450 brands of shoes and that nearly 200 of them are prefaced with "Dr." Other parts deal with hallux, affections of the nails, of the skin, of the bones. A good chapter discusses the way in which constitutional diseases affect the feet,—it should be noted that they emphasize the importance of a general examination. The technic of strapping and foot exercises wind up the book.

The book is printed in large pleasing type on dull paper, as is so often the case with Lippincott publications.

THE DIAGNOSIS AND TREATMENT OF DISEASES OF THE ESOPHAGUS. By PORTER P. VINSON, B. S., M. A., M. D., D. Sc., F. A. C. P., Professor of Bronchoscopy, Medical College of Virginia, Richmond, Virginia. 224 pages, with 98 illustrations. Price, $4. Springfield and Baltimore: Charles C Thomas, Publisher, 1939.

Many, many volumes have been devoted to the lower end of the alimentary tract; it is strange therefore that the upper end has just received its first accolade in a book devoted solely to the esophagus. In other books diseases of this vital tube have been considered along with lesions of the larynx, trachea and bronchi, and approach to their management is from the viewpoint of the endoscopist rather than the internist. Vinson shows that often the diagnosis can be made from the history, oftener from visualizing the esophagus with barium; at the same time of course he does not minimize visual inspection.

In his discussion of the "so-called Plummer-Vinson syndrome" he notes the exfoliation at the angles of the mouth: it is a bit surprising that he does not suggest that this cheilosis is due to riboflavin deficiency.

Probably every practitioner should have access to this book. The thoracic surgeon, the endoscopist and the gastroenterologist each needs a copy as a part of his armamentarium.

This book is "dedicated to the memory of the late Henry S. Plummer, a rare genius, who provided much of the impetus for the present satisfactory management of lesions of the esophagus." This dedication serves to endear it to another devoted student of Dr. Plummer's.

INJECTION TREATMENT OF HERNIA, HYDROCELE, GANGLION, HEMORRHOIDS, PROSTATE GLAND, ANGIOMA, VARICOCELE, VARICOSE VEINS, BURSAE, AND JOINTS. By PENN RIDDLE, B.S., M.D., F.A.C.S., Assistant Professor of Clinical and Operative Surgery, Baylor University, College of Medicine; Director of the Varicose Vein Clinic, Parkland Hospital, Dallas, Texas. 290 pages, with 153 illustrations. Price, $5.50. Philadelphia and London: W. B. Saunders Company, 1940.

Four years ago, not without considerable trepidation, the Editor of The Southern Surgeon published a paper by C. O. Rice and L. M. Larson on the injection treatment of hernia. One of these authors was a personal friend, a man whose honesty and integrity were beyond question, so the Editor pioneered in accepting the paper for publication. He did so in spite of the fact that at that time most surgeons frowned on any approach to the problem of hernia other than surgical. It is therefore with relief and pride that he has watched the growing recognition of this mode of therapy and he welcomes the more this book on injection treatment.

Riddle makes plain it requires no less knowledge of anatomy, no less surgical acumen and skill to treat a hernia by injection than by the well

established surgical procedures. He also makes plain that not all cases are suitable for injection. Even in the most skilful hands, surgery carries a mortality of about 0.5 per cent and the percentage of cures is distinctly less than 100. Injection carries no mortality, in properly selected cases gives a percentage of cures almost as high as in those treated surgically, and there is no period of hospitalization. He states that, in the event injection does not give a perfect result, subsequent operation is more satisfactorily performed.

The sections on varicose veins and hemorrhoids are adequate, but perhaps no better than other discussions of these subjects.

The injection of mercurochrome into the prostate does not appeal to this reader. However when the author states that, after having each vas exposed and 5 c.c. of mercurochrome injected into it, "many men" in Grant's series "later became fathers," it arouses skepticism, even though it be proved their wives later became mothers.

Riddle has produced a splendid exposition of the present status of injection treatment and one that, with a few possible exceptions, is not colored by unchecked enthusiasm. It is well worth attention.

SYPHILIS AND ITS ACCOMPLICES IN MISCHIEF: SOCIETY, THE STATE AND THE PHYSICIAN. By GEORGE M. KATSAINOS, M. D. 672 pages. Privately printed at Athens, Greece, by Kyklos Publishing Company, 1939.

This fat tome contains a great deal of interest: it is rich in quotations from the classics and it presents many fascinating items in the history of syphilis. Its author is evidently a learned man. He expresses his opinions freely on a wide variety of subjects. Of course he has a perfect right to express his opinions as freely as he desires even when they run counter to the consensus of American authors. At the same time we have a perfect right not to read his opinions. Our principal objection to this book is its verbosity: it rambles all over the map. It was privately printed and therefore it appears exactly as Dr. Katsainos wanted it: had it been gotten out by a well established publishing house, the blue pencil would have been used freely and the reader could have more easily familiarized himself with Dr. Katsainos's ideas as to syphilis.

SCIENTIFIC EXHIBITS

Dr. H. Earle Conwell, Chairman

Continuous movies from 9:30 a. m. to 4:00 p. m., showing certain surgical technics, pathology and treatment. Scientific exhibits will be shown on the Mezzanine floor of the Tutwiler Hotel. The movies will be in the Pine Room on the second floor.

Drs. A. Ochsner and Michael DeBakey.....*Carcinoma of the Lung Surgery of the Sympathetics*

Dr. Dean H. Echols.....*Ruptured Intervertebral Disc—A Cause of Sciatic Pain*

Drs. Meadows and Kesmodel.....*Bone Tumors*

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Dr. Warren A. Coleman.....*Movie—Surgical Accidents: Their Results, Causes and Prevention*

Drs. C. S. Venable and Walter Stuck....*Application of Vitallium Cap in Arthroplasty of the Hip*

Dr. J. P. Robertson.....*Cystoscopic Findings in Children with Persistent Pyuria*

Drs. Willard Bartlett and R. W. Bartlett..*Original Studies in Goiter*

Drs. R. H. Rigdon and Paul F. Stookey..*Observation on Staphylococci and Staphylococci Toxin*

Dr. H. A. Davis.....*Pathology of Surgical Shock*

Drs. J. M. Mason, Lloyd Noland and R. M. Pool..*Vascular Injuries and Disease*

Dr. John L. Keeley.....*Physiological Changes in Experimental Burns*

Dr. Chalmers H. Moore.....*Neurosurgical Cases—Clinic of Dr. Chalmers H. Moore*

Dr. John Duff.....*Pyelotomy for Calculus*

Dr. C. N. Carraway.....*Pentothal Sodium Anaesthesia*

Drs. Alton Ochsner and Howard R. Mahorner....*Modern Treatment of Varicose Veins*

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The Southern Surgeon is now published monthly. Subscription price in the United States and Canada: $5.00; in other countries: $6.00, including postage. Single Copies, $1.00 postpaid.

Checks may be made payable to The Southern Surgeon Publishing Co.